1859-
1959
100
YEARS OF
SERVICE

THE Journal OF THE Kansas Medical Society

NOVEMBER
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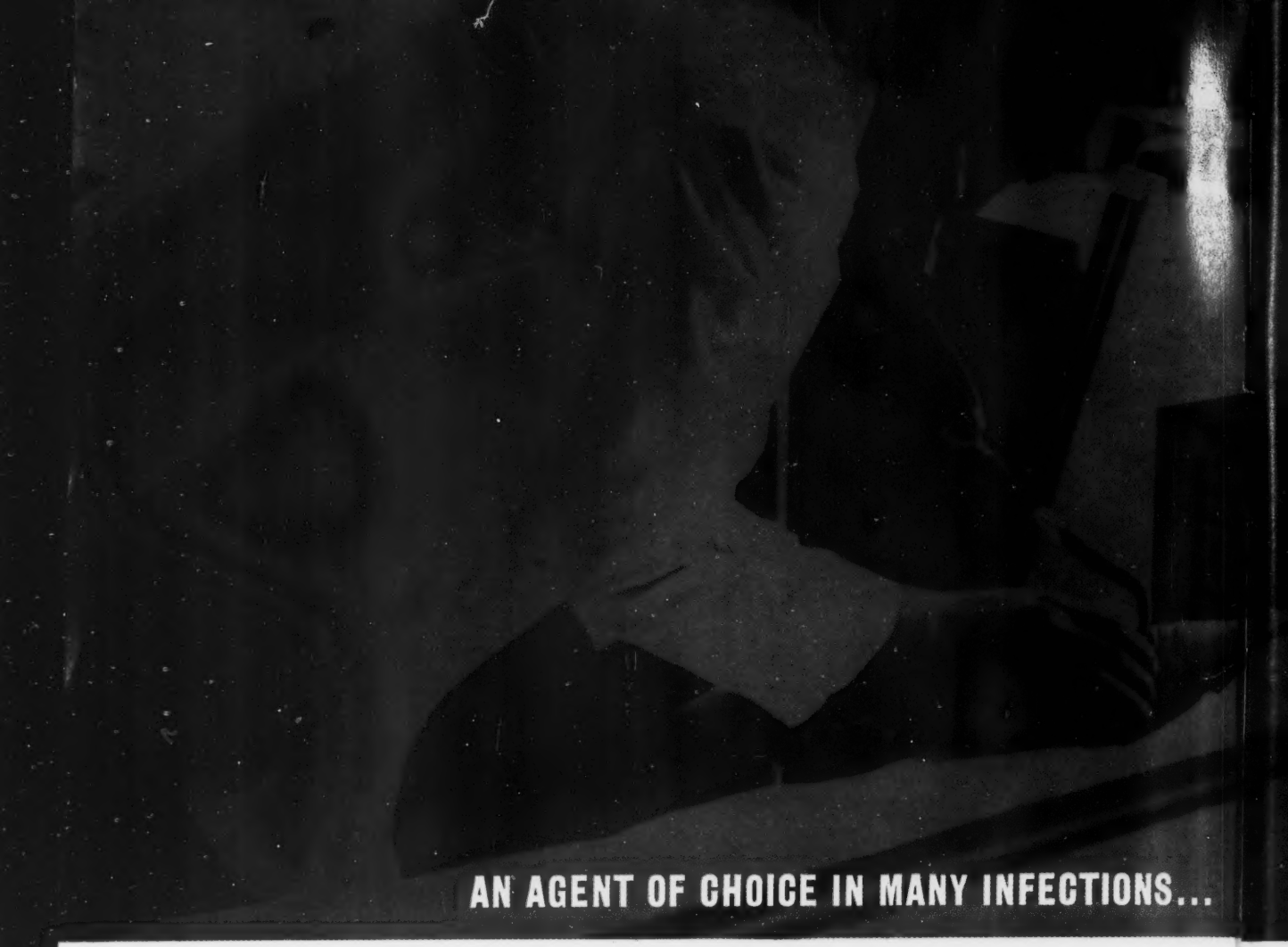
VOL. LX
NO. XII



AN AGENT OF CHOICE IN MANY INFECTIONS...

CHLOROMYCETIN®

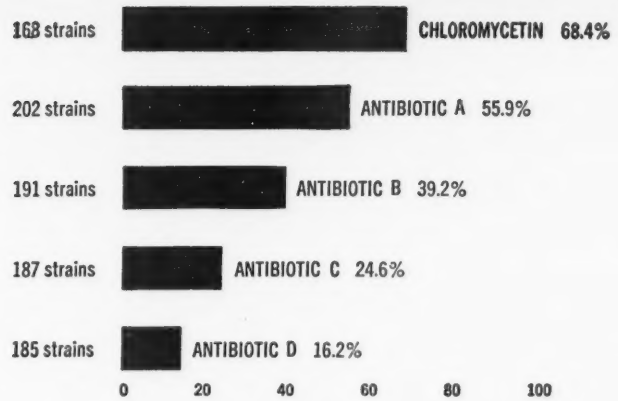
"In selecting the antibiotic of choice for treating urinary pathogens, *in vitro* testing is essential."[1] Numerous studies[2-9] attest the wide antibacterial activity of CHLOROMYCETIN—"...often effective against organisms which are resistant to the other broad-spectrum antibiotics."[3] For example: "...it often provides a means of controlling infections due to such resistant organisms as *Proteus*."[3] "*B. proteus* exhibits a greater sensitivity to chloramphenicol than to other antibiotics," according to one investigator.[4] Another reported: "*Proteus* bacilli are often drug resistant, but significant activity against them is exhibited by chloramphenicol...."[5] In the latter study, CHLOROMYCETIN "...showed the greatest activity among the agents tested against *E. coli, A. aerogenes,* and *Proteus* species."[5]

CHLOROMYCETIN (chloramphenicol, Parke-Davis) is available in a variety of forms, including Kapseals® of 250 mg. in bottles of 16 and 100.

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with i administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain oth drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

REFERENCES: (1) Holloway, W. J., & Scott, E. G.: *Delaware M. J.* **29**:159, 1957. (2) Suter, L. S., & Ulrich, E. W.: *Antibiotic & Chemother.* **9**:38, 1959. (3) Murphy, J. J., & Rattner, W. H.: *J.A.M.A.* **166**:616, 1958. (4) Rhoads, P. S.: *Postgrad. Med.* **21**:563, 1957. (5) Horton, B. E., & Knight, V.: *J. Tennessee M. A.* **48**:367, 1955. (6) Seneca, H.: *Am. Pract. & Digest Treat.* **10**:622, 1959. (7) Hill, W. H.: *M. Clin. North America* **43**:191, 1959. (8) Seneca, H., *et al.*: *J. Urol.* **81**:324, 1959. (9) Wolfsohn, A. W.: *Connecticut Med.* **22**:769, 1958.

IN VITRO SENSITIVITY OF PROTEUS SPECIES TO CHLOROMYCETIN AND TO FOUR OTHER ANTIBIOTICS*



*Adapted from Suter & Ulrich.[2]
These antibiotics were tested by the tube dilution method, using a concentration of 12.5 mcg/ml. The percentages represent the total number of sensitive strains found in five *Proteus* species.

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89059

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Volume LX NOVEMBER, 1959 Number 12

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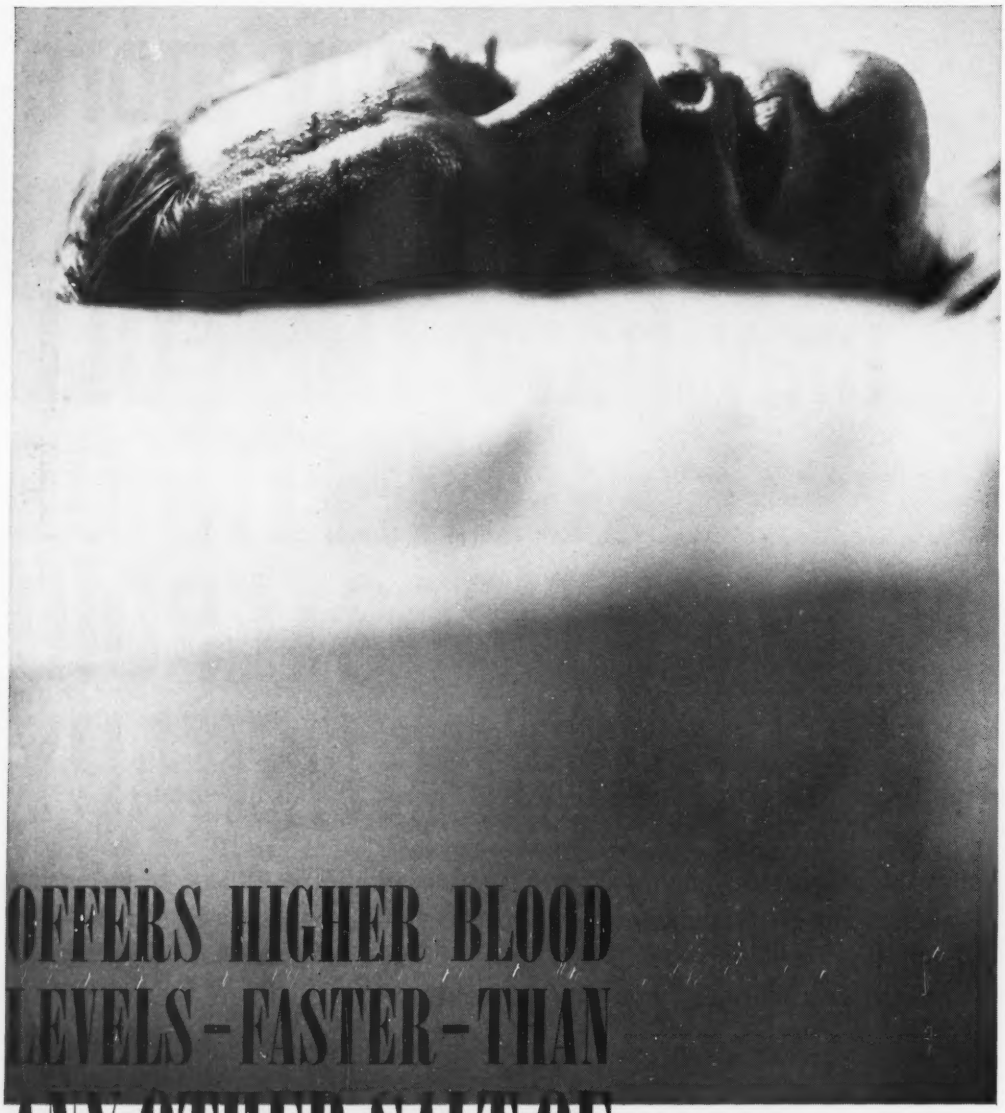

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for the tense and nervous patient

relief comes fast and comfortably

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TRAUMATIC STRAINS AND BRUISES

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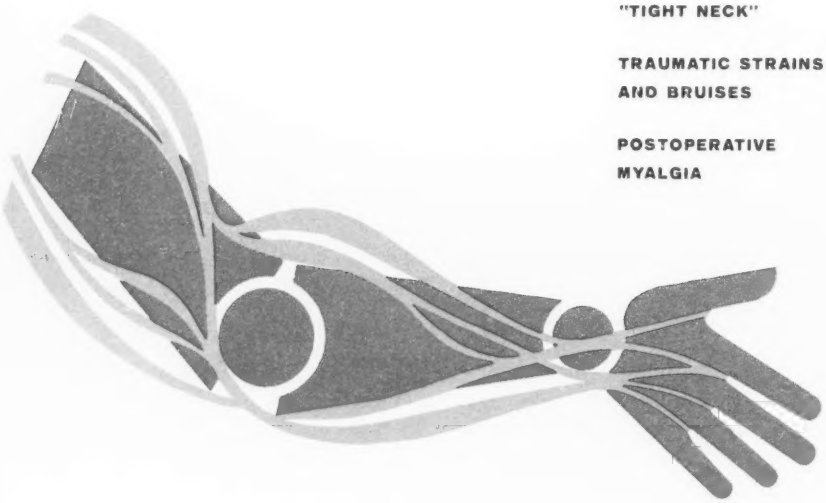

■ Exhibits unusual analgesic properties, different from those of any other drug ■ Specific and superior in relief of SOMAtic pain ■ Modifies central perception of pain without abolishing natural defense reflexes ■ Relaxes abnormal tension of skeletal muscle

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■ More specific than salicylates ■ Less drastic than steroids

■ More effective than muscle relaxants

SOMA has an unique analgesic action. It apparently modifies central pain perception without abolishing peripheral pain reflexes. SOMA is particularly effective in relieving joint pain. Patients say that they feel better and sleep better with SOMA than with previously used analgesic, sedative or relaxant drugs.
SOMA also relaxes muscle hypertonia, with its stresses on related joints, ligaments and skeletal structures.

ACTS FAST. Pain-relieving and relaxant effects start in 30 minutes and last 6 hours.

NOTABLY SAFE. Toxicity of SOMA is extremely low. No effects on liver, endocrine system, blood pressure, blood picture or urine have been reported. Some patients may become sleepy, particularly on high dosage.

EASY TO USE. Usual adult dose is one 350 mg. tablet 3 times daily and at bedtime.

SUPPLIED: Bottles of 50 white coated 350 mg. tablets.

Literature and samples on request.

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NIAMID*

reduces pain in angina pectoris

NIAMID, in intensive clinical tests, has proved to have a high degree of safety and to be a valuable adjunct in the management of the anginal syndrome. NIAMID produces striking symptomatic improvement in angina patients...

- reduces frequency of anginal episodes
- diminishes severity of attacks
- decreases nitroglycerin requirements
- renews sense of well-being

Note: Because of dramatic relief of symptoms and increased sense of well-being in anginal cases, it is advisable to caution the patient against overexertion.

DOSAGE: Start with 75 mg. of NIAMID daily in single or divided doses. After 2 weeks or more, adjust the dosage, depending upon patient response, in steps of one or one-half 25 mg. tablet. Once improvement is seen, gradually reduce dosage to the maintenance level. Many patients respond to NIAMID within a few days, others within 7 to 14 days. NIAMID is available as 25 mg. (pink) and 100 mg. (orange) scored tablets.

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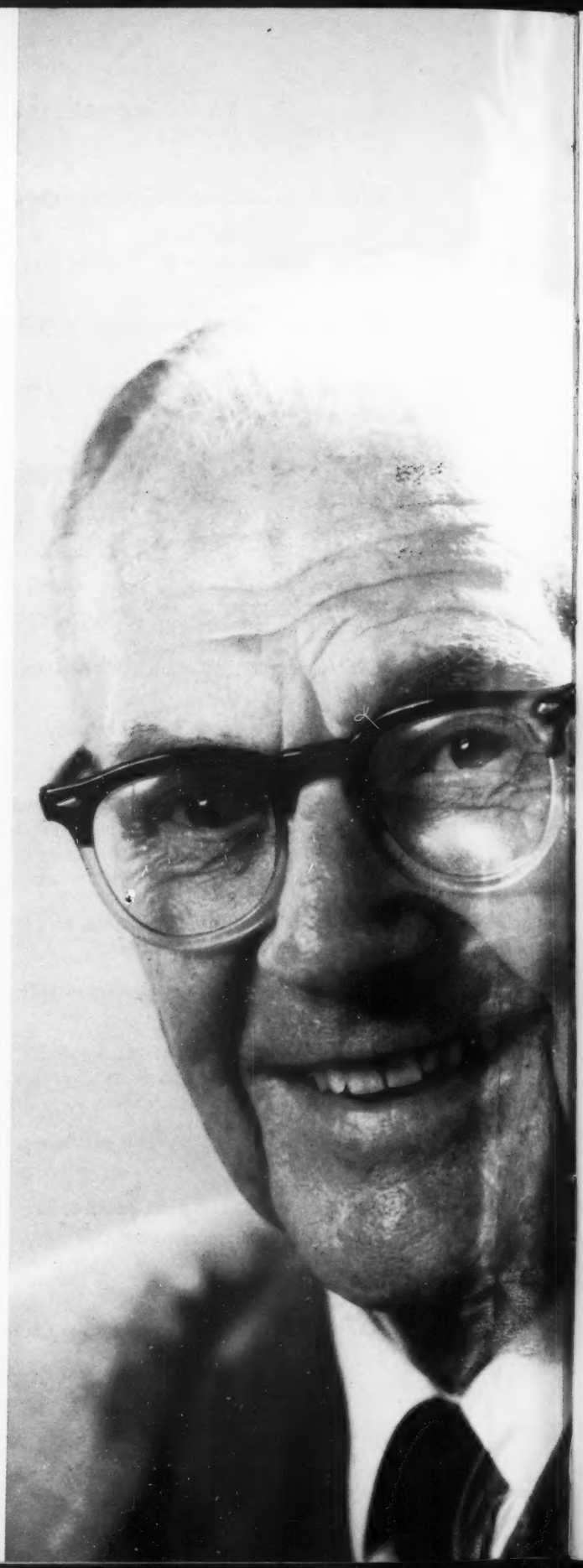

internal and/or external attack

Whatever the bacterial infection seen in EENT, the foci respond rapidly to a suitable form of broad-spectrum ACHROMYCIN. In superficial cases, local therapy is often dramatic. In deep-seated conditions, ACHROMYCIN V capsules complement topical control for fast relief and remission.

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Ophthalmic Ointment 1%
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e Ear Solution

n Nasal Suspension with Hydrocortisone And Phenylephrine

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ACHROMYCIN V (Tetracycline with Citric Acid) Capsules

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running noses and open stuffed noses orally

Triaminic®

the leading oral nasal decongestant

- in nasal and paranasal congestion
- in sinusitis
- in postnasal drip
- in allergic reactions of the upper respiratory tract.

safer and more effective than topical medication[1,2,3]

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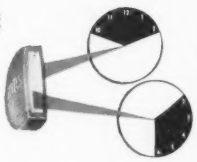
Relief with Triaminic is prompt and prolonged because of this special timed-release action . . . beneficial effect starts in minutes, lasts for hours



first — the outer layer dissolves within minutes to produce 3 to 4 hours of relief

then — the core disintegrates to give 3 to 4 *more* hours of relief

Each TRIAMINIC Tablet provides:
Phenylpropanolamine HCl .....50 mg.
Pheniramine maleate .....25 mg.
Pyrilamine maleate .....25 mg.

One-half of this formula is in the outer layer, the other half is in the core.

Dosage: One tablet in the morning, mid-afternoon and at bedtime.

References: 1. Lhotka, F. M.: Illinois M. J. 112: 259 (Dec.) 1957. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958.

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NEW PINEAPPLE FLAVOR Overwhelmingly selected by a taste panel.

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DOSAGE: Three to five troches daily for three to five days.

SUPPLIED: Vials of 12.

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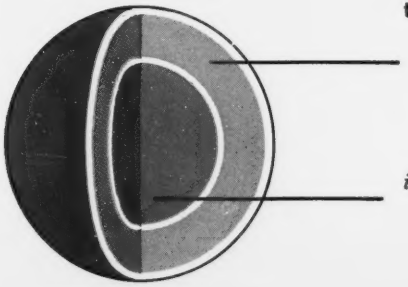
the complaint: "nervous indigestion"

the diagnosis: any one of several nonspecific gastrointestinal disorders requiring relief of symptoms by sedative-antispasmodic action with concomitant digestive enzyme therapy. **the prescription:** a new formulation, incorporating in a single tablet the actions of Donnatal and Entozyme. **the dosage:** two tablets three times a day, or as indicated.



the formula: *in the gastric-soluble outer layer:*

Hyoscyamine sulfate	0.0518 mg.
Atropine sulfate	0.0097 mg.
Hyoscine hydrobromide	0.0033 mg.
Phenobarbital (⅛ gr.)	8.1 mg.
Pepsin, N.F.	150 mg.

in the enteric-coated core:

Pancreatin, N.F.	300 mg.
Bile salts	150 mg.

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For Complete Formula see PDR (Physicians' Desk Reference), page 689

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Lederle

QUESTIONS ON THE CLINICAL USE OF KANTREX®

Q Why should I use KANTREX® Injection* when there are so many other antibiotics available?

A

Because KANTREX Injection is bactericidal to a wide variety of organisms, including many that are highly resistant to the other antibiotics[3,4,10,12,13,17,18,20,21,23,24,25,27,30,33,35,37]

—organisms such as *Staph. aureus, Staph. albus, A. aerogenes, E. coli, H. pertussis, K. pneumoniae, Neisseria* sp., *Shigella, Salmonella* and many strains of *B. proteus.*

Q *But if I use* KANTREX *Injection, won't that help make bacteria resistant to it also?*

Next page, please .....

* **Kanamycin sulfate injection (Bristol)**

Q But if I use KANTREX *Injection, won't that help make bacteria resistant to it also?*

A A very good question, but it is reassuring to note that in almost two years of clinical use of KANTREX for the treatment of infections for which it is recommended, the emergence of KANTREX-resistant bacterial populations has not been a problem.

Q My impression is that KANTREX *is just another neomycin. Isn't that so?*

A Indeed not. The only thing KANTREX and neomycin have in common is a similar antimicrobial spectrum. Otherwise, they're very different: they have different chemical structures; the toxicity of KANTREX is "much less than that of neomycin"[14]; and clinically, KANTREX Injection is practical for systemic administration routinely, while neomycin is not.

Q You mean that KANTREX *Injection doesn't have the nephrotoxicity of neomycin?*

A Precisely. It's true that when KANTREX Injection is used, urinary casts – even slight albuminuria or microscopic hematuria – may appear, especially in poorly hydrated patients, but this does not reflect any progressive damage to the kidneys. These signs promptly disappear on adequate hydration or termination of therapy.

Q Then why do you recommend reduced dosage in patients with renal impairment?

A Because renal impairment causes an excessive accumulation of KANTREX in the blood and tissues, when usual doses are administered. Since KANTREX Injection is excreted entirely by the kidneys, renal impairment leads

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to unnecessarily high and prolonged blood levels; and such excessive concentrations increase the risk of ototoxicity.

Q *Is that why we see reports of patients developing hearing loss during* KANTREX *Injection therapy?*

A Yes. A study of the few reported cases in which patients have suffered impaired hearing will show that in every instance they had pre-existing or concurrent renal impairment, yet received usual or excessive doses of KANTREX Injection. Dosage recommendations for KANTREX Injection emphasize that in patients with renal dysfunction, adequate serum levels can be achieved with a fraction of the dose suggested for patients with normal kidney function – with minimal risk of ototoxicity.

Q *Since urinary tract infections are often accompanied by renal impairment, does that mean I shouldn't use* KANTREX *Injection in such conditions?*

A Not at all. With proper precautions, KANTREX Injection is an excellent drug for the treatment of urinary tract infections, especially those due to *Proteus, A. aerogenes* and *E. coli*, even when renal impairment is present.

Q *What are the "proper precautions" in a patient with impaired renal function?*

A The package literature covers them in detail. First, the daily dose should be reduced in such a patient. Then, if he is going to receive KANTREX Injection for 7 days or more, a pre-treatment audiogram should be done, and it should be repeated at appropriate intervals during therapy. If tinnitus or subjective hearing loss develops, or if followup audiograms show significant loss of high frequency response, KANTREX therapy should be discontinued. However, therapy for 7 days or more

is seldom required because the clinical response to KANTREX Injection is so rapid.

Q *Why do you put so much emphasis on* KANTREX*'s "rapid action"? Every antibiotic I've heard about is supposed to be "rapid acting."*

A There is such an abundance of clinical evidence about "rapid acting" that it takes KANTREX Injection out of the "supposed-to" class.[1,2,3,7,8,9,11,15,16,19,21,22,26,29,32,33] Remember, the effectiveness of KANTREX Injection therapy can usually be appraised in 24 to 36 hours. That's definite evidence of rapid action. In fact, one group of investigators reported that "the rapidity with which bacteria are killed by this agent is reflected by the promptness of the clinical response."[29]

Q *Does* KANTREX *Injection cause blood dyscrasias?*

A In extensive clinical and toxicity studies by numerous investigators, as well as almost two years of general use, not a single instance of such toxicity has been reported.

Q *Can I administer* KANTREX *Injection in any other way than by the intramuscular route?*

A Yes. While it's usually given intramuscularly, other routes are practicable: intravenous, intraperitoneal, by aerosol, and as an irrigating solution. Complete instructions are included in the package insert.

Q *So you think I ought to use* KANTREX *Injection as my first choice antibiotic in staph and gram-negative infections?*

A Yes – because all evidence to date indicates that it is bactericidal against a wide range of organisms... rapid acting... does not encourage development of bacterial resistance... is well tolerated in specified dosage... and has not caused any blood dyscrasias.

for local gastrointestinal therapy... not for systemic infections

Q *Why can't I use* KANTREX *Capsules for systemic medication?*

A Because there is only negligible absorption of KANTREX from the gastrointestinal tract.[3,5,6,8,28,34] Thus, capsules cannot provide effective blood levels.

Q *Then what are* KANTREX *Capsules used for?*

A Preoperative bowel sterilization, and local treatment of intestinal infections due to kanamycin-sensitive organisms.

Q *I've been using neomycin for preoperative bowel sterilization. Why should I switch to* KANTREX *Capsules?*

A Because KANTREX has been rated as "superior to neomycin" for this purpose.[6] It provides rapid and satisfactory control of coliforms, clostridia, staphylococci and streptococci; yeasts do not proliferate; stool concentrations of the drug are exceptionally high; and nausea, vomiting or intestinal irritation have not been observed.[5,6]

Q *What advantages do* KANTREX *Capsules offer me in the treatment of intestinal infections?*

A A high degree of effectiveness against most of the pathogens responsible for such infections: *Salmonella, Shigella, Staph. aureus, E. coli* and *Endamoeba histolytica.* Moreover, their use has been "remarkably free of any side effects."[31]

INJECTION KANAMYCIN SULFATE INJECTION

INDICATIONS

Infections due to kanamycin-sensitive organisms, particularly staph or "gram-negatives": genito-urinary infections; skin, soft tissue and post-surgical infections; respiratory tract infections; septicemia and bacteremia; osteomyelitis and periostitis.

DOSAGE: INTRAMUSCULAR ROUTE

Recommended daily dose is 15 mg. per kg. of body weight, in 2 to 4 divided doses.

For intramuscular administration, KANTREX Injection should be injected deeply into the upper outer quadrant of the gluteal muscle.

TOXICITY

When the recommended precautions are followed, the incidence of toxic reactions to KANTREX is low. In well hydrated patients under 45 years of age with normal kidney function, receiving a total dose of 20 Gm. or less of KANTREX, the risk of ototoxic reactions is negligible.

In patients with renal disease and impaired renal function, the daily dose of KANTREX should be reduced in proportion to the degree of impairment to avoid accumulation of the drug in serum and tissues, thus minimizing the possibility of ototoxicity. In such patients, if therapy is expected to last 7 days or more, audiograms should be obtained prior to and during treatment. KANTREX therapy should be stopped if tinnitus or subjective hearing loss develops, or if audiograms show significant loss of high frequency response.

OTHER ROUTES OF ADMINISTRATION

KANTREX should be used by intravenous infusion only when the intramuscular route is impracticable. KANTREX can also be employed for intraperitoneal use, aerosol treatment, and as an irrigating solution. See package insert for directions.

PRECAUTIONS

Use of antibiotics may occasionally result in overgrowth of non-sensitive organisms. If superinfection appears during therapy, appropriate measures should be taken.

SUPPLY

Available in rubber-capped vials as a ready-to-use sterile aqueous solution in two concentrations (stable at room temperature indefinitely):

KANTREX Injection, 0.5 Gm. kanamycin (as sulfate) in 2 ml. volume.
KANTREX Injection, 1.0 Gm. kanamycin (as sulfate) in 3 ml. volume.

CAPSULES (for local gastrointestinal therapy; not for systemic medication)

INDICATIONS AND DOSAGE

For preoperative bowel sterilization: 1.0 Gm. (2 capsules) every hour for 4 hours, followed by 1.0 Gm. (2 capsules) every 6 hours for 36 to 72 hours.

For intestinal infections: Adults: 3.0 to 4.0 Gm. (6 to 8 capsules) per day in divided doses for 5 to 7 days. Infants and children: 50 mg. per kg. per day in 4 to 6 divided doses for 5 to 7 days.

PRECAUTION

Preoperative use of KANTREX Capsules is contraindicated in the presence of intestinal obstruction. Although only negligible amounts of KANTREX are absorbed through intact intestinal mucosa, the possibility of increased absorption from ulcerated or denuded areas should be considered.

SUPPLY

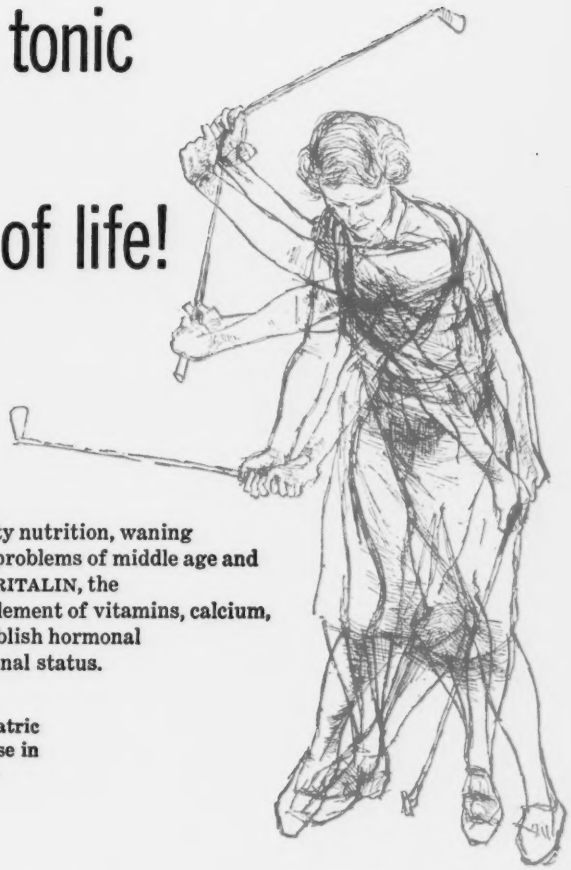
KANTREX Capsules, 0.5 Gm. kanamycin (as sulfate), bottles of 20 and 100.

REFERENCES:

1. Andrieu, G., Monnier, J., and Bourse, R.: Presse Med. 67:718, 1959. 2. Berger, S. H., and Wehrle, P. F.: Ann. N. Y. Acad. Sci. 76:136, 1958. 3. Bunn, P. A., Baltch, A., and Krajnyak, O.: *Ibid.* 76:109, 1958. 4. Bunn, P. A., and Baltch, A.: New Eng. J. Med. 259:659, 1958. 5. Cohn, I., Jr., and Longacre, A. B.: S. G. & O. 108:100, 1959. 6. Cohn, I., Jr.: Ann. N. Y. Acad. Sci. 76:212, 1958. 7. Cronk, G. A., and Naumann, D. E.: *Ibid.* 76:308, 1958. 8. Cronk, G. A., and Naumann, D. E.: J. Lab. & Clin. Med. 53:888, 1959. 9. Davies, F. G.: Ann. N. Y. Acad. Sci. 76:129, 1958. 10. Dougherty, L. J., Seneca, H., and Lattimer, J. K.: Antibiotics Annual 1958-1959, p. 713. 11. Dube, A. H.: Am. Pract. & Digest Treat. 10:1165, 1959. 12. Finegold, S. M., *et al.*: Antibiotics Annual 1958-1959, p. 606. 13. Finegold, S. M., *et al.*: Ann. N. Y. Acad. Sci. 76:319, 1958. 14. Finland, M.: *Ibid.* 76:391, 1958. 15. Finland, M.: Lancet 2:209, 1958. 16. Fujii, R., *et al.*: J. Japan M. A. 39:740, 1958; abst. World-Wide Abst. Gen. Med. 2:28, 1959. 17. Gourevitch, A., Hunt, G. A., and Lein, J.: Ant. & Chemo. 8:149, 1958. 18. Gourevitch, A., *et al.*: Ann. N. Y. Acad. Sci. 76:31, 1958. 19. Greey, P. H., and Wightman, K. J. R.: *Ibid.* 76:224, 1958. 20. Griffith, L. J., and Ostrander, W. E.: Ant. & Chemo. 9:416, 1959. 21. Hewitt, W. L., and Finegold, S. M.: Ann. N. Y. Acad. Sci. 76:122, 1958. 22. High, R. H., Sarria, A., and Huang, N. N.: *Ibid.* 76:289, 1958. 23. Hirsh, H. L.: Sibley Mem. Hosp. Alumni Assn. Bull. 2:16, 1959. 24. Hunt, G. A., and Moses, A. J.: Ann. N. Y. Acad. Sci. 76:81, 1958. 25. Lattimer, J. K., Seneca, H., Zinsser, H. H. and Troc, O.: J.A.M.A. 170:938, 1959. 26. Prigot, A., Shidlovsky, B. A., and Campbell, E. A.: Ann. N. Y. Acad. Sci. 76:204, 1958. 27. Riley, H. D., Jr.: Antibiotics Annual 1958-1959, p. 623. 28. Ruiz Sanchez, F., *et al.*: *Ibid.*, p. 725. 29. Rutenburg, A. M., Koota, G. M., and Schweinburg, F. B.: Ann. N. Y. Acad. Sci. 76:348, 1958. 30. Slotkin, E. A.: Postgrad. Med. 25:433, 1959. 31. Thurman, W. G., and Platou, R. V.: Ann. N. Y. Acad. Sci. 76:230, 1959. 32. Welch, H., *et al.*: *Ibid.* 76:66, 1958. 33. White, A., and Knight, V.: *Ibid.* 76:277, 1958. 34. Yow, E. M.: Practitioner, 182:759, 1959. 35. Yow, E. M., *et al.*: A.M.A. Arch. Int. Med. 102:948, 1958. 36. Yow, M. D., Desmond, M. M., and Nickey, L. N.: J. Ped. 54:409, 1959. 37. Yow, M. D., and Womack, G. K.: Ann. N. Y. Acad. Sci. 76:363, 1958.

Bristol LABORATORIES INC. • Syracuse, New York

New revitalizing tonic brightens the second half of life!



Ritonic™

A sense of frustration and inadequacy, faulty nutrition, waning gonadal function—RITONIC meets all these problems of middle age and senile let-down. The unique combination of RITALIN, the *safe* central stimulant, with a balanced complement of vitamins, calcium, and hormones acts to renew vitality, re-establish hormonal and anabolic benefits, and improve nutritional status.

"We found Ritonic to be a safe, effective geriatric supplement . . ."[1] "Patients reported an increase in alertness, vitality and sense of well being."[2]

PRESCRIBE RITONIC

for your geriatric patients, your middle-aged patients and your postmenopausal patients.

Each Ritonic Capsule contains:

Ritalin® hydrochloride	5 mg.
methyltestosterone	1.25 mg.
ethinyl estradiol	5 micrograms
thiamin (vitamin B_1)	5 mg.
riboflavin (vitamin B_2)	1 mg.
pyridoxin (vitamin B_6)	2 mg.
vitamin B_{12} activity	2 micrograms
nicotinamide	25 mg.
dicalcium phosphate	250 mg.

Remember
SERPASIL®
(reserpine CIBA)
for the anxious hypertensive with or without tachycardia

Dosage: One Ritonic Capsule in mid-morning and one in mid-afternoon.
Supplied: Ritonic CAPSULES; bottles of 100.
References: 1. Natenshon, A. L.: J. Am. Geriatrics Soc. *6*: 534 (July) 1958.
2. Bachrach, S.: To be published.
RITALIN® hydrochloride (methylphenidate hydrochloride CIBA)

CIBA SUMMIT, N. J.

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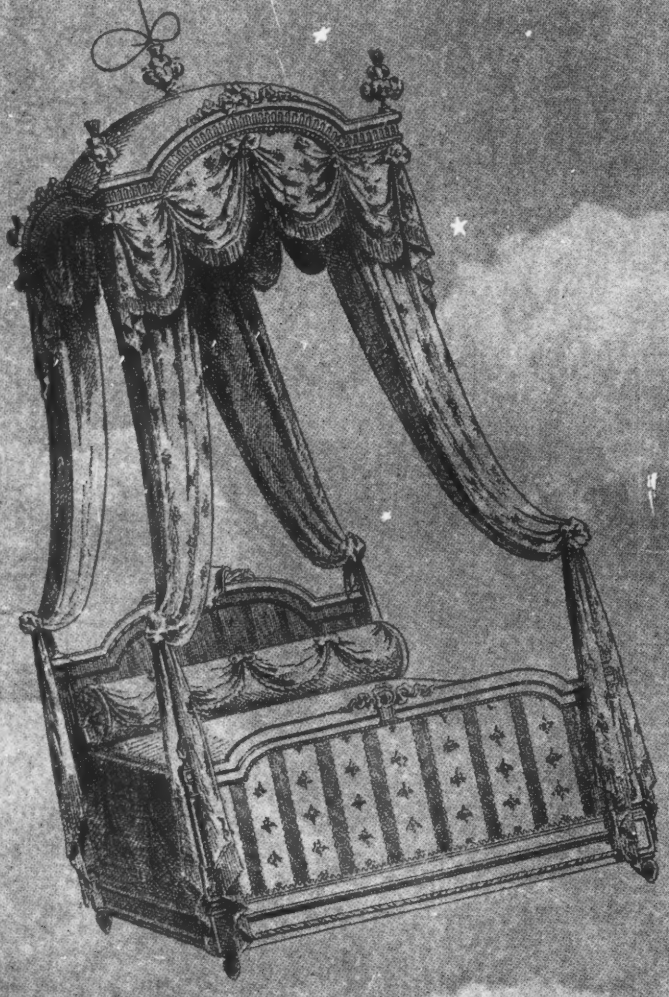
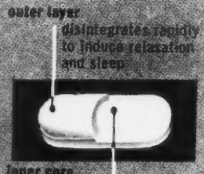
For those patients who complai

carries your patients through the middle of the night





Each Nebralin timed-release tablet contains:

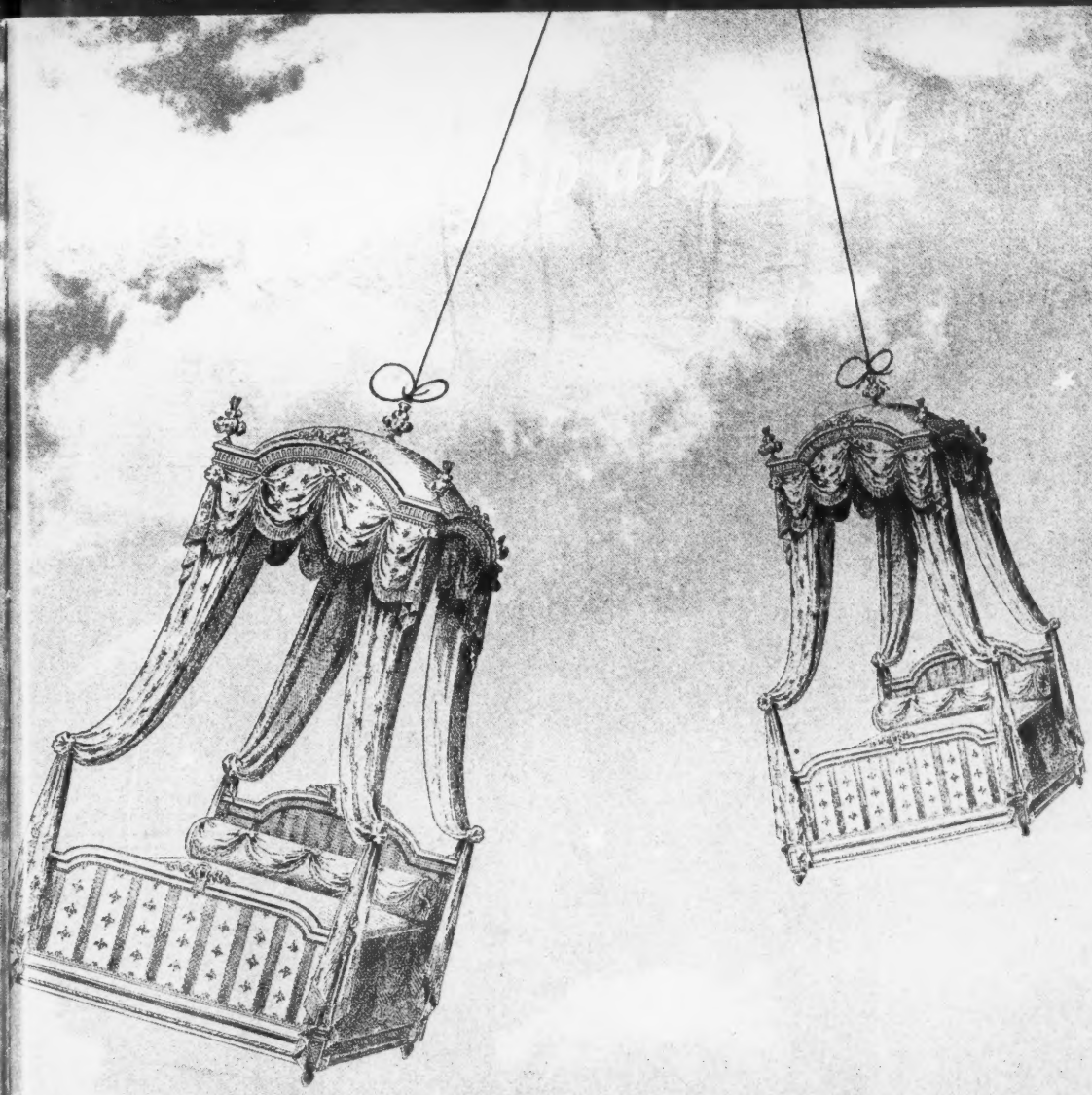
Dorsital* ..... 90 m
Warning: May be habit forming

Mephenesin ..... 425 m

*Dorsey brand of pentobarbital

CAUTION: Federal law prohibits dispensing without prescription

Dosage: One or two tablets ½ h before retiring.



nebralin®

timed-release tablet

timed-release action for a full night's sleep

NEBRALIN is designed to duplicate the normal sleep pattern. It encourages muscular relaxation and induces sustained, relaxed sleep by the release of Dorsital and mephenesin in a timed-release tablet. Rapid-acting mephenesin quickly relaxes skeletal muscles to overcome "fatigue-tension" and conditions the body for sleep. Dorsital provides CNS sedation to induce sound, relaxed sleep. The initial and sustaining dosages are designed to keep the amount of barbiturate to be inactivated at any one time at a low level tapering toward morning. Evidence indicates that mephenesin is capable of producing sleep,[1] and when combined with a barbiturate enhances barbiturate action.[2,3] Moreover, the integrated action of the two components permits smaller dosages of each,[4] assuring your patients refreshed awakenings without "morning hangover."

1 Schlesinger, E. B.: Tr. New York Acad. Sc. 2:6, (Nov.) 1948.
2 Richards, R. K., and Taylor, J. D.: Anesthesiology 17:414, 1956.
3 Shideman, F. E.: Postgrad. Med. 24:207, 1958.
4 Berger, F.: Pharmacol. Rev. 1:243, 1949.

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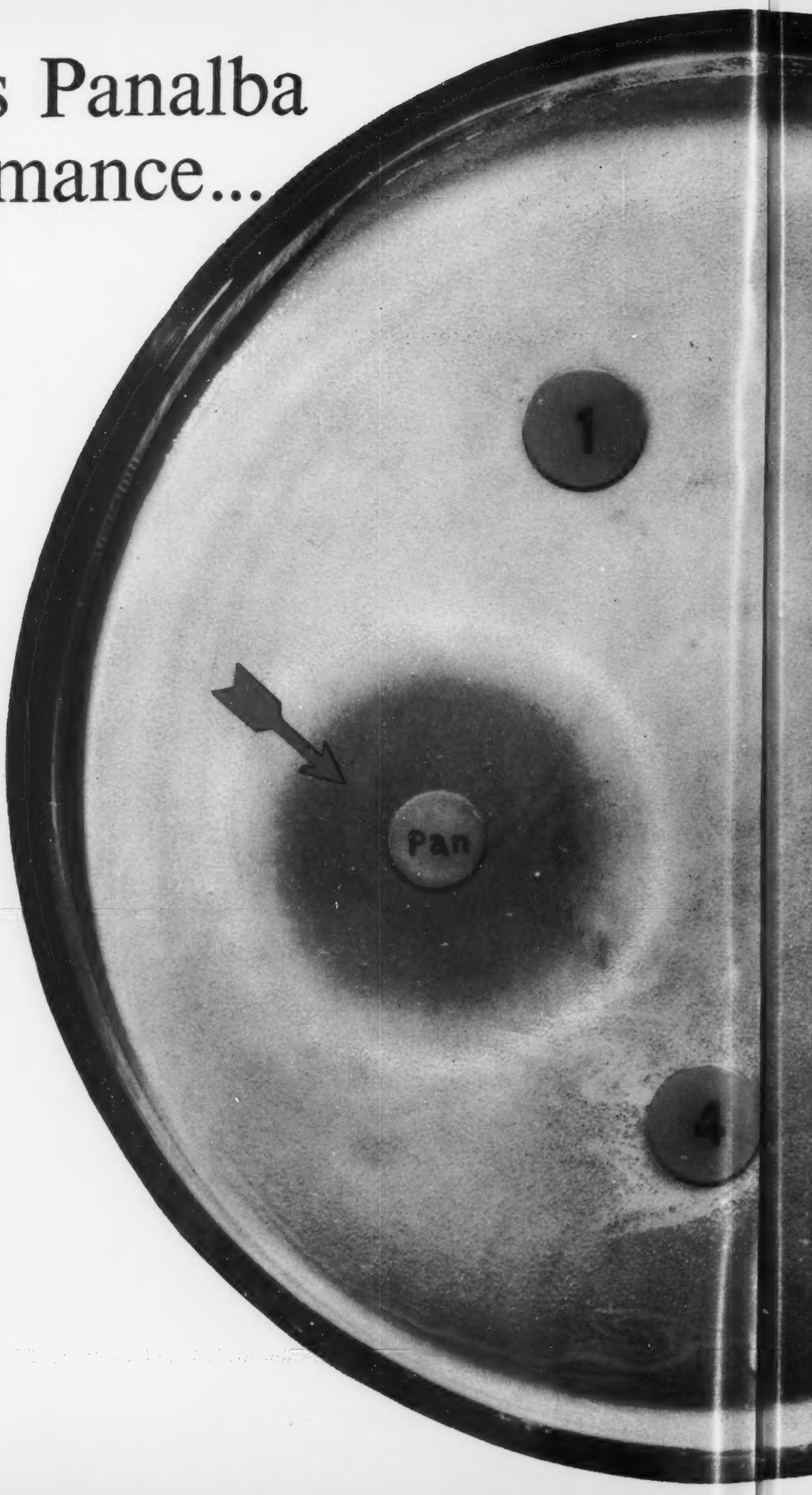
This is Panalba performance...



in pneumonia

. . . into a mixed culture of the three organisms commonly involved in pneumonia . . . *K. pneumoniae*, *Diplococcus pneumoniae*, and *Staphylococcus aureus* (in this case a resistant strain) . . . we introduce the five most frequently used antibiotics.

Twenty-four hours later (in this greatly enlarged photograph), note that only *one* of the five leading antibiotics has stopped *all* the organisms, including the resistant staph! This is Panalba.

In your next pneumonia patient . . . in *all* your patients with potentially-serious infections . . . provide this extra protection with your prescription:

Dosage—1 or 2 capsules 3 or 4 times a day. Supplied—Capsules containing Panmycin phosphate equivalent to 250 mg. tetracycline hydrochloride, and 125 mg. Albamycin as novobiocin sodium, in bottles of 16 and 100. *Now available:* new Panalba Half-Strength Capsules in bottles of 16 and 100.

Panalba*

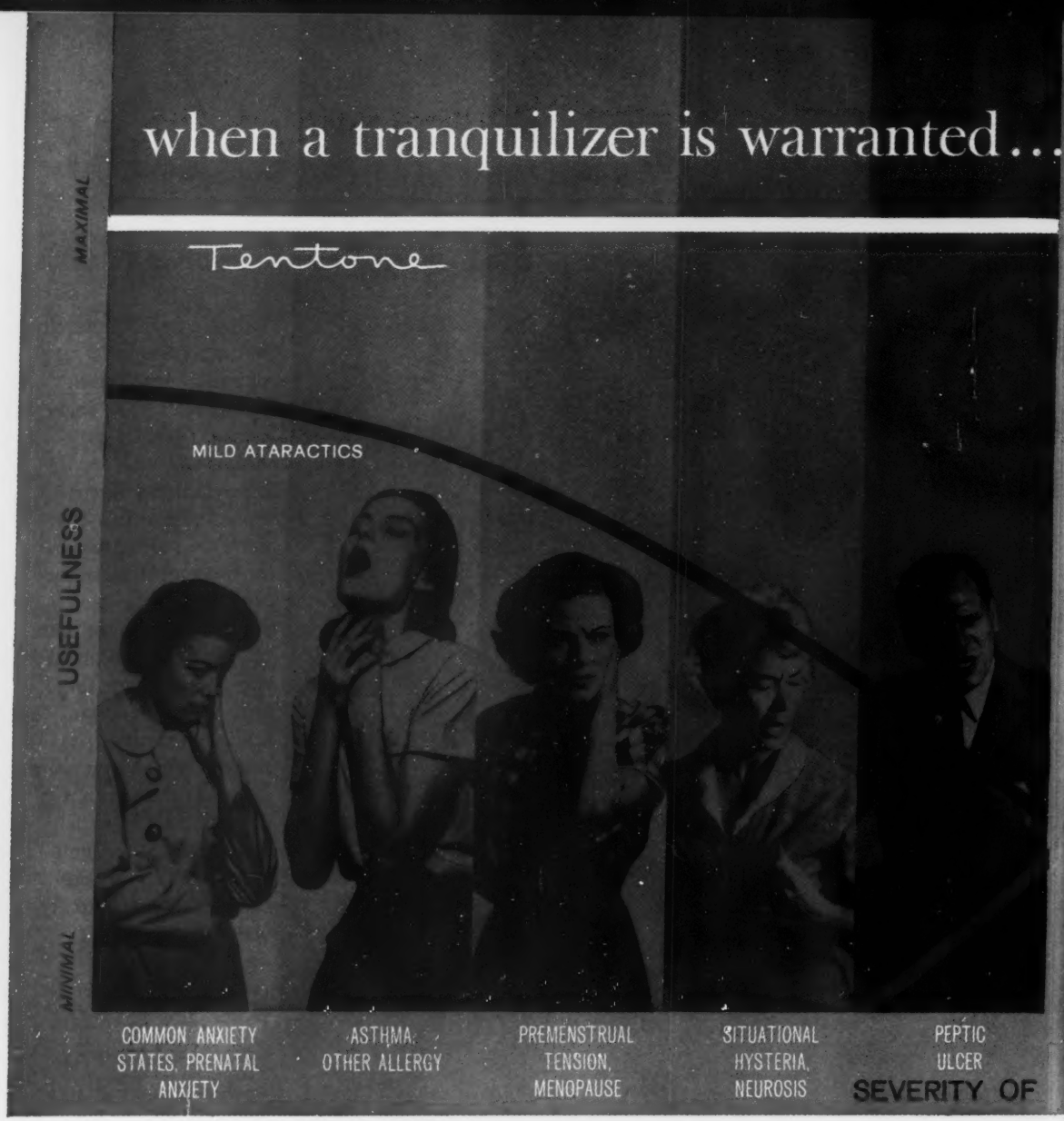
(Panmycin* Phosphate plus Albamycin*)

The broad-spectrum antibiotic of *first* resort

Upjohn The Upjohn Company Kalamazoo, Michigan

*TRADEMARK, REG. U. S. PAT. OFF.

when a tranquilizer is warranted...



The extended usefulness of TENTONE is readily apparent

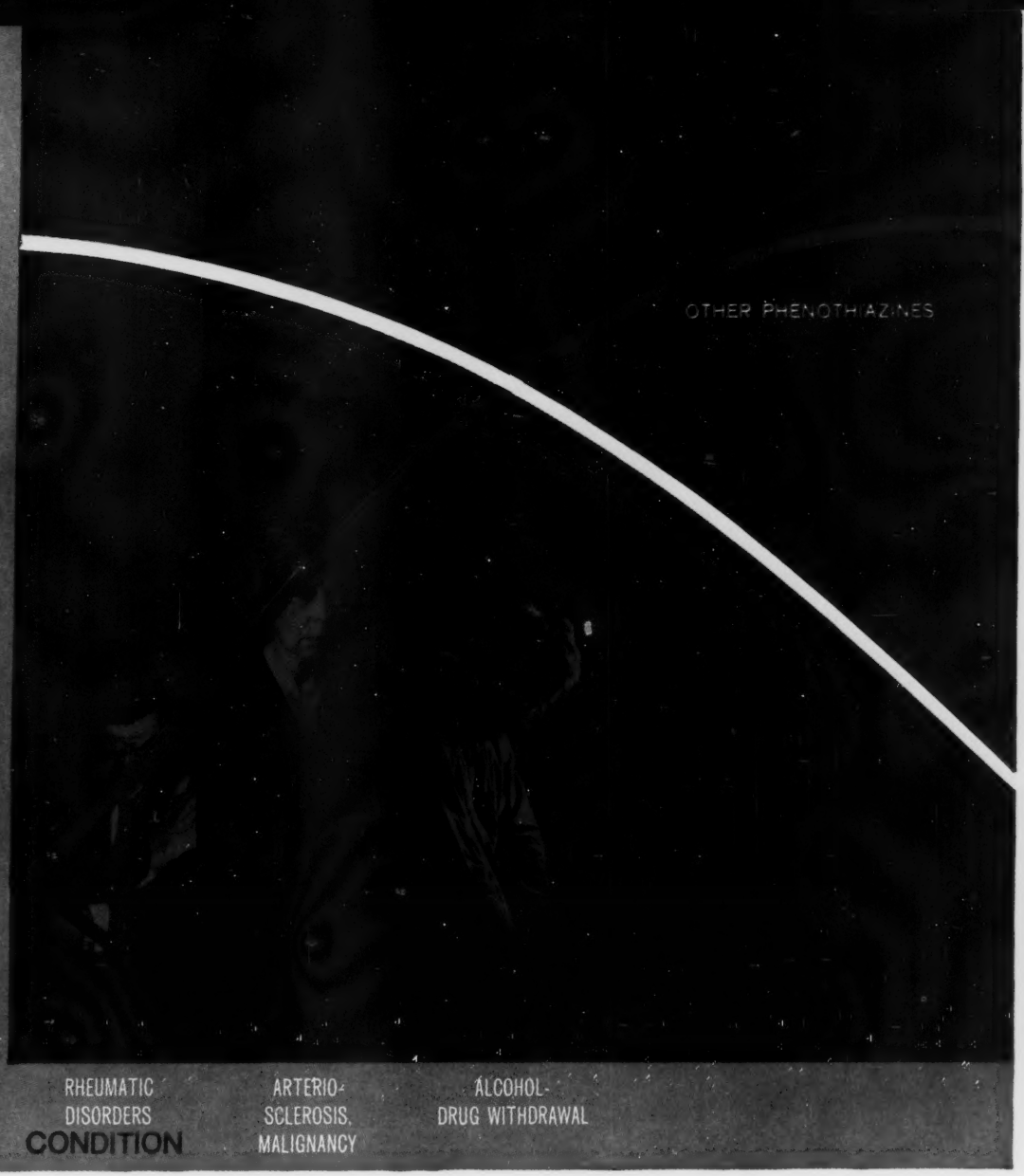
TENTONE® Methoxypromazine Maleate is a new, distinctive phenothiazine... highly active ...for general use in mild and moderate emotional and psychosomatic disorders.

TENTONE elicits a striking, positive calming response[1,2]...with marked reduction of psychic disorientation, and low risk of blood, liver or other organic toxicity and intolerance.[1-4]

TENTONE parallels the weaker ataractics in low incidence of side effects. Freedom from induced depression is apparently even greater.[5]

TENTONE provides a broadly adaptable dosage range (30 to 500 mg. daily) to permit maximum control in cases of varying severity.

TENTONE is also indicated to relieve emotional stress in surgical, obstetric and other hospitalized patients.



Dosage: Mild to moderate cases—average starting dose, one 10 mg. or one 25 mg. tablet three or four times daily. *Moderate to severe*—average starting dose, one 50 mg. tablet four times daily. *Supplied:* 10 mg., 25 mg., and 50 mg. tablets.

1. Bodi, T., and Levy, H.: Clinical report, cited with permission. 2. Wetzler, R. A., and Phillips, R. M.: Clinical report, cited with permission. 3. Prigot, A.: Clinical report, cited with permission. 4. Gosline, E., *et al.*: *Am. J. Psychiat.* 115:939 (April) 1959. 5. Turvey, S. E. C.: Clinical report, cited with permission.

Tentone

Methoxypromazine Maleate

Lederle

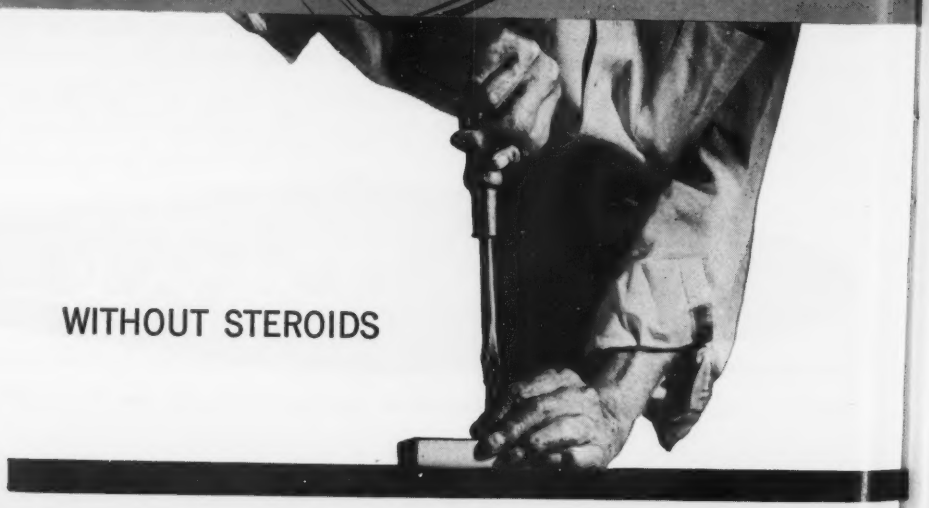
LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

WITHOUT STEROIDS

In every arthritic state...

WITHOUT STEROIDS

WITHOUT STEROIDS



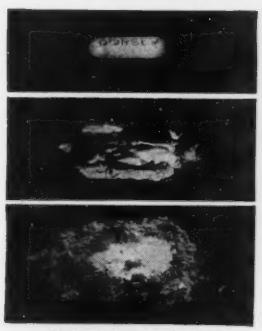
MAINTENANCE THERAPY WITHOUT STEROIDS IS FUNDAMENTAL

Sound, conservative therapy with salicylates has been consistently reaffirmed as basic, long-term maintenance therapy in the arthritides.

Buffered Pabirin provides superior maintenance therapy. It epitomizes fundamental long-term basic therapy since it can be given month after month without serious complications and with minimal problems to patient and doctor alike.

Buffered Pabirin is formulated to provide high and sustained salicylate blood levels. Each tablet consists of an outer layer containing a buffer (aluminum hydroxide), para-aminobenzoic acid, and ascorbic acid; a core of acetylsalicylic acid.

In the stomach, the outer layer quickly releases the buffer, which protects against nausea, dyspepsia and other gastrointestinal symptoms so frequently encountered with salicylates alone. The core of Buffered Pabirin then disintegrates rapidly, permitting rapid absorption of the acetylsalicylic acid for faster pain relief.



Photographs show 2-stage Tandem Release disintegration.

Each tablet contains:

Acetylsalicylic acid (5 gr.)	300 mg.
Para-aminobenzoic acid (5 gr.)	300 mg.
Ascorbic acid	50 mg.
Dried aluminum hydroxide gel	100 mg.

All Buffered Pabirin is sodium- and potassium-free.

Dosage: Two or three tablets 3 or 4 times daily.

References: 1. Hart, D.; Bagnall, A. W.; Bunim, J. J., and Polley, F. H.: Ninth International Congress on Rheumatic Diseases, Toronto, Ont. (June 25) 1957. 2. Report of Joint Committee, Medical Research Council & Nuffield Foundation, Treatment of Rheumatoid Arthritis, British Medical Journal (April 13) 1957. 3. Friend, D. G.: New England J. Med. 257:278 (Aug.) 1957.

Buffered **Pabirin®** *Tablets*

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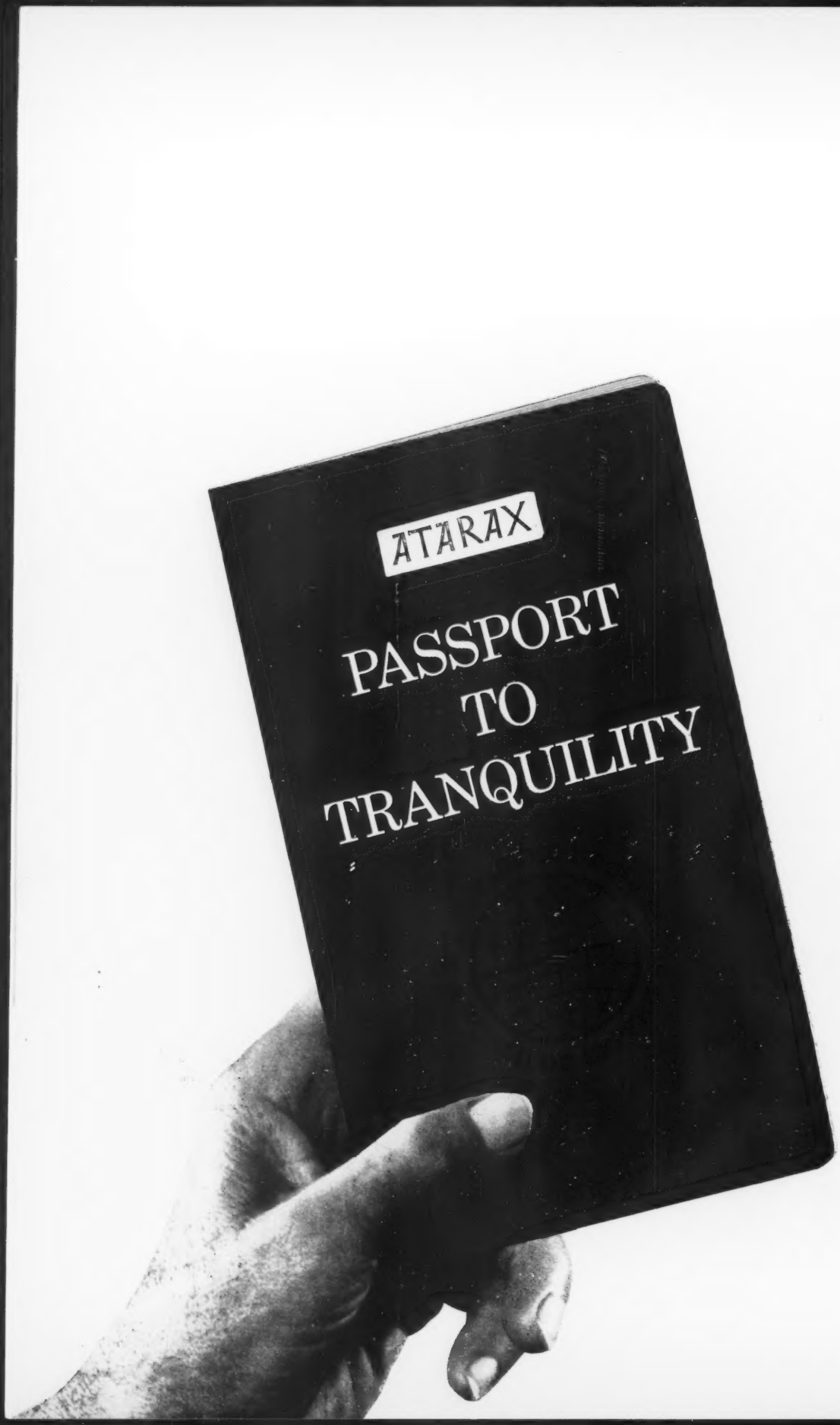
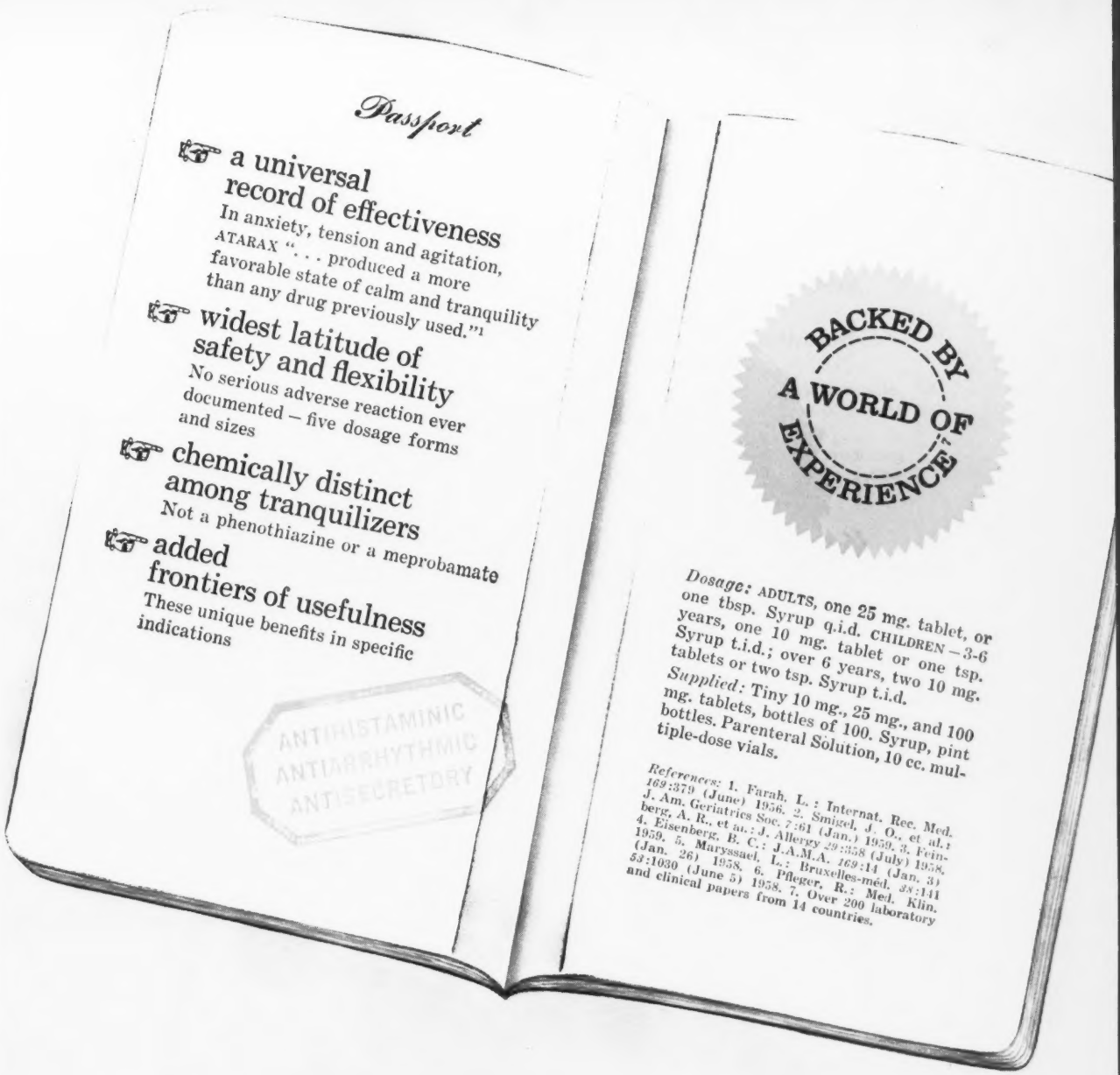

ATARAX
PASSPORT
TO
TRANQUILITY


Passport
a universal record of effectiveness
In anxiety, tension and agitation, ATARAX "... produced a more favorable state of calm and tranquility than any drug previously used."1
widest latitude of safety and flexibility
No serious adverse reaction ever documented – five dosage forms and sizes
chemically distinct among tranquilizers
Not a phenothiazine or a meprobamate
added frontiers of usefulness
These unique benefits in specific indications
ANTIHISTAMINIC
ANTIARRHYTHMIC
ANTISECRETORY
BACKED BY A WORLD OF EXPERIENCE
Dosage: ADULTS, one 25 mg. tablet, or one tbsp. Syrup q.i.d. CHILDREN – 3-6 years, one 10 mg. tablet or one tsp. Syrup t.i.d.; over 6 years, two 10 mg. tablets or two tsp. Syrup t.i.d.
Supplied: Tiny 10 mg., 25 mg., and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.
References: 1. Farah, L.: Internat. Rec. Med. 169:379 (June) 1956. 2. Smigel, J. O., et al.: J. Am. Geriatrics Soc. 7:61 (Jan.) 1959. 3. Feinberg, A. R., et al.: J. Allergy 29:358 (July) 1958. 4. Eisenberg, B. C.: J.A.M.A. 169:14 (Jan. 3) 1959. 5. Maryssael, L.: Bruxelles-med. 38:141 (Jan. 26) 1958. 6. Pfleger, R.: Med. Klin. 53:1030 (June 5) 1958. 7. Over 200 laboratory and clinical papers from 14 countries.

ATARAX®

(brand of hydroxyzine)

PASSPORT TO TRANQUILITY

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

relieve the tension—and control its G.I. sequelae

...Pathibamate®

meprobamate with PATHILON® tridihexethyl chloride Lederle

for relieving tension and curbing hypermotility and excessive secretion in G. I. disorders

PATHIBAMATE combines two highly effective and well-tolerated therapeutic agents:

meprobamate (400 mg. or 200 mg.)—a tranquilizer and muscle-relaxant widely accepted for the effective management of tension and anxiety

PATHILON (25 mg.)—an anticholinergic long noted for producing prompt symptomatic relief through peripheral, atropine-like action, yet with few side effects

now available...

PATHIBAMATE-200 Tablets

200 mg. meprobamate • 25 mg. PATHILON

for more flexible control of G. I. trauma and tension smooth, sugar-coated, easy-to-swallow

PATHIBAMATE-400 and PATHIBAMATE-200 are indicated for duodenal ulcer; gastric ulcer; intestinal colic; spastic and irritable colon; ileitis; esophageal spasm; anxiety neurosis with gastrointestinal symptoms and gastric hypermotility.

Supplied: **PATHIBAMATE-400**—Each tablet (yellow, 1/2-scored) contains meprobamate, 400 mg.; PATHILON tridihexethyl chloride 25 mg.

PATHIBAMATE-200—Each tablet (yellow, coated) contains meprobamate, 200 mg.; PATHILON tridihexethyl chloride, 25 mg.

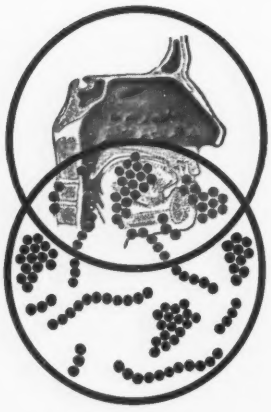
Administration and Dosage: **PATHIBAMATE-400**—1 tablet three times a day at mealtime and 2 tablets at bedtime.

PATHIBAMATE-200—1 or 2 tablets three times a day at mealtime and 2 tablets at bedtime.

Adjust dosage to patient response.

Contraindications: glaucoma; pyloric obstruction, and obstruction of the urinary bladder neck.

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York



when upper respiratory congestion is complicated by bacterial invaders

TRISULFAMINIC provides logical therapy

for the patient ill with congestion and infection of the upper respiratory tract, as in purulent rhinitis, sinusitis, tonsillitis and otitis media, when caused by sulfa-susceptible bacteria;

• because secondary invasion by such bacteria so frequently follows the common cold.[1]

the reasons for combining Triaminic with triple sulfas

Triaminic and triple sulfas are not only pharmacologically *compatible*, they are a therapeutically *logical* combination for upper respiratory infections: Triaminic for effective decongestant relief from rhinitis, rhinorrhea and sinusitis;[2] triple sulfas for well-established antibacterial action.

The advantages of Trisulfaminic in upper respiratory infections include: proved effectiveness; safety; economy; ease of administration; less likelihood of sensitivity reactions;[3] compatibility with antibiotics and other antibacterial therapy. Provided also as Suspension for additional convenience.

Trisulfaminic®

TRIAMINIC WITH TRIPLE SULFAS

Available as TABLETS and SUSPENSION

Each easy-to-swallow Trisulfaminic Tablet or 5 ml. teaspoonful of Suspension provides:

Triaminic® .....25 mg.
(phenylpropanolamine HCl 12.5 mg.
pheniramine maleate ..... 6.25 mg.
pyrilamine maleate ..... 6.25 mg.)
Trisulfapyrimidines, U.S.P. .....0.5 Gm.

Dosage:

Adults—2 to 4 tablets or tsp. initially, followed by 2 tablets or tsp. every 4 to 6 hours until the patient has been afebrile 3 days. *Children 8 to 12* — 2 tablets or tsp. initially, followed by 1 tablet or tsp. every 6 hours. *Children under 8*—dosage according to weight.

The palatability, convenience and effectiveness of the Suspension make it especially suitable for children and for those older patients who prefer liquid medication.

References: 1. Cecil, R. L., et al.: J.A.M.A. 124:8 (Jan. 1) 1944. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Beckman, H.: Drugs, Their Nature, Action & Use, Saunders, Philadelphia, 1958, p. 527.

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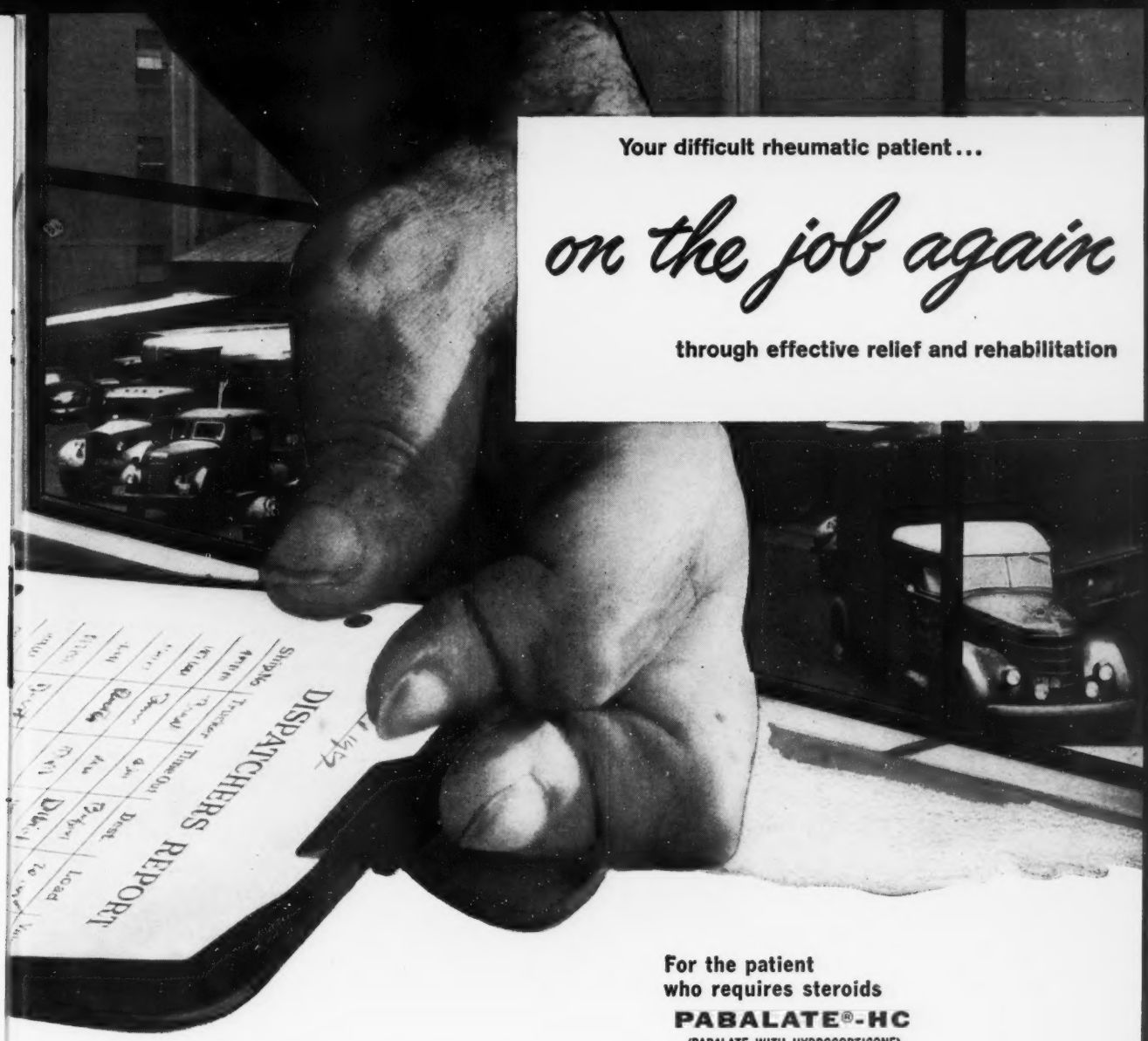


Your difficult rheumatic patient...

on the job again

through effective relief and rehabilitation

For the patient who does not require steroids

PABALATE®

Reciprocally acting nonsteroid antirheumatics . . . more effective than salicylate alone.

In each enteric-coated tablet:

Sodium salicylate U.S.P.....0.3 Gm. (5 gr.)
Sodium para-aminobenzoate .....0.3 Gm. (5 gr.)
Ascorbic acid .....50.0 mg.

or for the patient who should avoid sodium

PABALATE®-Sodium Free

Pabalate, with sodium salts replaced by potassium salts.

In each enteric-coated tablet:

Potassium salicylate .....0.3 Gm. (5 gr.)
Potassium para-aminobenzoate .....0.3 Gm. (5 gr.)
Ascorbic acid .....50.0 mg.

For the patient who requires steroids

PABALATE®-HC

(PABALATE WITH HYDROCORTISONE)

Comprehensive synergistic combination of steroid and nonsteroid antirheumatics... full hormone effects on low hormone dosage . . . satisfactory remission of rheumatic symptoms in 85% of patients tested.

In each enteric-coated tablet:

Hydrocortisone (alcohol) ..... 2.5 mg.
Potassium salicylate ..... 0.3 Gm.
Potassium para-aminobenzoate.. 0.3 Gm.
Ascorbic acid .....50.0 mg.

PABALATE® Robins PABALATE®-HC

For steroid or non-steroid therapy: SAFE DEPENDABLE ECONOMICAL

A. H. ROBINS CO., INC., RICHMOND 20, VIRGINIA • Ethical Pharmaceuticals of Merit since 1878

RESEARCH:

key to Kent's popularity

In 1958, Kent made the greatest gain in popularity ever recorded by any filter cigarette in any year—a sales increase of 20-billion cigarettes.

Behind this popularity is a story of months and years of research, perfecting the remarkable combination of filter action and flavor found in today's Kent cigarette. In developing Kent, Lorillard research scientists recognized that smokers wanted, on the one hand, a really satisfying taste; on the other, reduced tars and nicotine. In addition, smokers demanded a free and easy draw.

These, then, were the objectives. The first scientific breakthrough in the project was the development of the exclusive Micronite filter, patented by Lorillard. This filter was created because of newly-discovered principles in the field of filtration, which have been previously described in these pages.

Though this filter satisfied everyone on its ability to reduce tars and nicotine to the lowest level among the largest selling brands, there was still work to be done in the areas of taste and draw. After additional months of research, a new tobacco blend was developed which delivered rich taste *after* the smoke had passed through the filter. Next in the series of laboratory triumphs was a method of improving the draw to compare with the most free-drawing of all filter brands.

The rest of the Kent story is a legend in the tobacco industry. Outside, independent research studies confirmed the fact that Kent had achieved its objectives. Smokers responded. In fact, during the past year, more smokers changed to Kent than to any other cigarette in America.

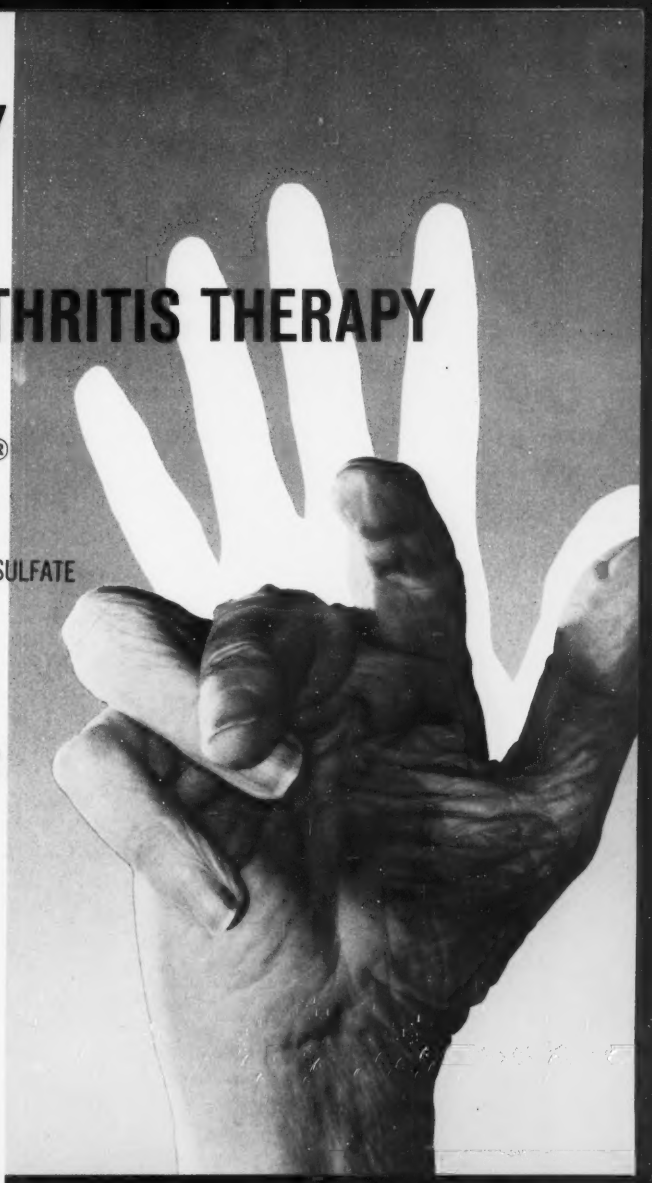
A Product of P. Lorillard Company—First with the finest cigarettes—through Lorillard Research!

MAINSTAY

OF RHEUMATOID ARTHRITIS THERAPY

Plaquenil® SULFATE

Brand of hydroxychloroquine sulfate



New Long Term Chemotherapy of RHEUMATOID ARTHRITIS

"Whatever else may be needed from time to time in the management of individual cases, these drugs [Plaquenil and Aralen] should always be given a prolonged trial (at least six months) as the 'mainstay' of therapy."

Bagnall, A. W. (Univ. British Columbia, Vancouver, B.C.): A.M.A. Clinical Meeting (Scientific Section, Exhibit No. 124), Minneapolis, Minnesota, Dec. 2-5, 1958.

"The 4-aminoquinoline drugs (Plaquenil and Aralen) together with supplemental agents administered in nontoxic doses effectively maintained suppression of the disease in 83 per cent of 194 patients followed for 18 months."

Scherbel, A. L.; Harrison, J. W., and Atdjian, Martin: **Cleveland Clin. Quart. *25:95, April, 1958.***

"When used in tolerated dosage and over a sufficient period of time, there appears to be a tremendous therapeutic potential in the antimalarial drugs. . . . Plaquenil in this study did not have as many side effects as Aralen and thus appears to be a more practical compound."

Cramer, Quentin (Kansas City): **Missouri Med. *55:1203, Nov., 1958.***

Plaquenil is the hydroxy derivative of Aralen® and is available as Plaquenil sulfate in tablets of 200 mg. (bottles of 100).

Average Dosage:

INITIAL—400 to 600 mg. (1 tablet 2 or 3 times daily)

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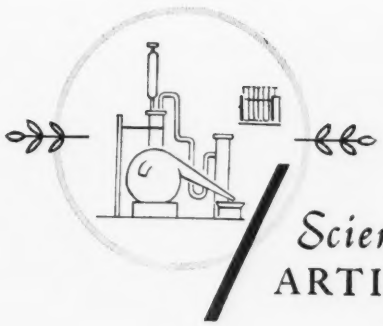

Scientific
ARTICLES

Active Treatment of Cerebral Palsy

A Report of the Work Being Conducted by the Institute of Logopedics, Wichita, Kansas

HENRY O. MARSH, M.D., ***Wichita***

Introduction

The Physicians of Kansas have shown a mounting interest in the program for the cerebral palsied at the Institute of Logopedics. The Institute of Logopedics is an institution in Wichita for the correction of the speech-handicapped, and has earned a national and international reputation for exceptional work in this field. Because almost all children with cerebral palsy have a speech handicap, a special clinic for them was established at the Institute in 1948.

In this decade, 427 patients have been examined in the cerebral palsy clinic. Seventy-two children are currently on the active treatment schedule in occupational therapy and physical therapy. The program for the habilitation of the cerebral palsied has been expanded constantly, and improved as experience has revealed more effective therapeutic methods as well as some previous misconceptions and errors. The comprehensive program now provides speech therapy, medical supervision (Orthopedic and Pediatric), physical and occupational therapy, special education, and socialization.

The purpose of this report is to answer the most frequent questions and to supply additional information for those interested in this difficult socio-medical problem. No attempt has been made to be analytical, but special emphasis has been placed on our particular therapeutic approach to the problem.

It should be emphasized that the cerebral palsied children represent a fraction of the patients at the Institute of Logopedics. Although the proportion of patients with cerebral palsy has increased steadily, the Institute of Logopedics exists for the correction of any speech handicaps, and the vast majority of the speech problems are unassociated with cerebral palsy.

Dr. Marsh is Director of Medical Services for Cerebral Palsy, Institute of Logopedics in Wichita, and Member of Orthopedic Staff, Wichita Clinic.

This article underscores the fact that in active treatment of Cerebral Palsy the Institute is dealing with complete human beings. A well-rounded program offering medical treatment, habilitation, speech training, education, and socialization is affording the optimum opportunity for these children to improve and be able to function in the everyday world.

The treatment of cerebral palsy has been undergoing a gradual change. This change is the result of the concentrated effort of many workers on a common problem. The common experience that resulted from confronting similar daily problems shifted the emphasis from passive to active treatment. The previous tenets of treatment were passive and consisted of (1) rhythm exercises practiced passively in time to nursery

rhymes, (2) relaxation practice with the exclusion of physical and mental stimuli, (3) full control bracing, and (4) little or no surgery.

The treatment of cerebral palsy at the Institute of Logopedics has advanced along more active lines during the past decade. The active concept of therapy has withstood the test in the arena of daily living and has been found to produce far superior results. This approach parallels contemporary theories of education. A child learns best by doing and by undergoing his own personal experiences. Therefore, our efforts are concentrated on the personal physical accomplishment at the child's actual level of performance. This initial accomplishment, no matter how minor, forms the springboard for better future performances.

In evaluating patients with cerebral palsy, therapy may be directed most effectively if one considers the major long-term issues, which are discussed in detail below.

Motivation

Motivation is the inner spark that starts the human engine producing the power and drive that culminates in a physical act or accomplishment. The establishment of motivation in the cerebral palsied is the key to the entire problem. The first spark of motivation must be carefully protected and fed the proper amounts of fuel. The child then will enjoy a continuing desire to perform increasingly difficult independent physical acts.

Failure to establish motivation usually wells from one of several sources: (1) The child may be too handicapped, mentally or physically, in which case nothing can be done; (2) The therapeutic team may fail to cope creatively with the situation; (3) The child may lack the innate ambition and drive to work hard enough and long enough to succeed; (4) Motivation may be stultified by a complete parental or custodial bankruptcy of insight.

Family (sociologic) handicaps may establish limiting barriers which block motivation. Favorable physical home surroundings and mental atmosphere may have never been created nor permitted to exist. The parents may have failed tragically to appraise the child's ability realistically. This invariably forces the child into one of two characteristic developmental patterns.

In one such unrealistic pattern, the parents' optimistic goals far exceed the child's capabilities. Sheer physical inadequacy then blocks each struggling attempt to achieve these goals, and denies the child the necessary sense of accomplishments, parental praise, and recognition for a successful performance. The antithesis of this situation is as common and just as fatal to physical improvement. The parent, guardian, or therapist anticipates and performs every physical act for the child which might be too difficult. This method is neat and quick. The child, with all his needs satisfied remains totally dependent, without effort, complacently willing and happy to be waited on like a king on a throne. Either pattern presents a difficult and challenging family situation that complicates the already difficult problem of motivating the cerebral palsied child.

The cerebral palsied child, regardless of his degree of handicap, learns by doing. Each accomplishment motivates higher level efforts. This motivational process follows logical steps just as one climbs a ladder beginning with the bottom rung. For example, head-balance and control is the lowest performance level on the ladder of physical habilitation. The practice and establishment of head-control requires individually fitted chairs that stabilize the body and extremities. These chairs impart a sense of bodily security which the child must have. The child is much more cooperative and workable once head-control is established. When the child achieves this first goal, he is motivated and able to go on to the next step, sitting-balance.

Individual therapy in an atmosphere of group activity is an excellent climate for the production of motivation. The child is stimulated in this situation to hold his head up so that he may observe the general fun. This is the basic prototype situation that can be created in a myriad of forms to stimulate the child's physical maturation. In this manner a constantly higher level of performance can be demanded and achieved.

Physical Therapy

Passive physical therapy has been largely discontinued and replaced by active physical therapy. The traditional passive therapeutic approach was conscientiously followed for several years and received a thorough clinical trial. The results were disillusioning. We were unable to relate any significant physical improvement to passive rhythm exercises, relaxation practice or full control bracing.

The full control bracing technique, which is employed in many clinics, proved to be unsatisfactory in our hands. Encased in these braces like a knight in shining armor a child can be grasped and walked about like a wooden soldier. This proved, in our clinic, to be a frustrating experience for both the child and therapist. The basic principles of body balance, the simple concept of foot thrust against the floor, or the necessity of downward hand pressure on canes or crutches were never mastered or understood by children who were fully braced. There was no adequate way to teach these principles. These simple physical concepts must become integral reflexes or walking is impossible.

Physical therapy at the Institute of Logopedics has evolved about the four following points: (1) active exercises for involved muscles, (2) stretching of tight or contracted muscles, (3) supervised daily practice with physical therapy equipment, and (4) most important of all, teaching in a logical progressive manner the increasingly difficult physical accomplishments. These points require further elucidation.

Muscular weakness may be due to a cerebral defect, or muscle weakness may be secondary to overstretching by an antagonistic hyperactive muscle. The hip abductor is the prime example of secondary muscle weakness. The gluteus medius is frequently overpowered by hyperactive hip adductors and becomes nonfunctional; it can become serviceable through active exercises. The muscles whose loss of power is primary to a cerebral defect can be partially restored in a similar fashion. Hours of repetitive muscle exercises are necessary to restore these muscle action patterns and the functional capacity. Passive, then assistive exercises are employed initially to give the feel and control of a specific muscle action. The acme of muscle power is produced with progressive resistance exercises.

Tight contracted muscles must be stretched or lengthened. This is fundamental. Muscles must have an effective functional length before work can be performed. Muscle stretching is carried out by the therapist or the parents on a supervised home program. The most frequently contracted muscles are the hip adductors, hamstrings, calf muscles, forearm pronators and the wrist flexors. Surgical tenotomies and myotomies may be necessary and inescapable, as manual stretching is not always successful.

In the supervised daily practice with the physical therapy equipment, hours are spent observing and working with the child in his efforts to move on an exercise mat, crawler or rolling chair. It may prove necessary to contrive a play situation that will motivate self-propulsion. The following play situations have proven repeatedly successful in stimulating self-propulsion.

TV an Aid

All of our children have seen and enjoyed the Western TV programs with Matt Dillon and Wyatt Earp. In acting out these programs the children take the part of the desperadoes and are placed in the town jail. This is done by drawing a chalk line on the floor about the crawler or rolling chair. The child breaks out of jail by propelling himself across the chalk line. This situation is highly effective in motivating self-propulsion, as all children are anxious to emulate their heroes.

A second situation which is highly effective utilizes a fort constructed from a large corrugated cardboard carton. This fort is placed on the floor completely surrounding the child except for one side which is the gate. The child must then propel himself through the gate if he is to see what the Indians are doing outside the fort. If there exists any degree of innate inquisitiveness or spirit of play, these situations work well.

A physical and mental mutation occurs when the child realizes that he, as an individual, can be self-propelled. This spark of self-motivated physical independence must be sustained through progressive physical stages to the highest attainable level of development. This will vary with the degree of handicap and may be independent walking, crutch or cane walking, propelling a wheel chair with the hands, or a rolling chair with the feet.

The training program is further promoted by all imaginable types of equipment. Crawlers, upright individual control chairs, rolling chairs to be pro-

pelled by the feet, special wheel chairs, and standing tables are but a few examples. The child's needs are the criteria and stimulus for the construction of some new piece of equipment. Each stage of physical development requires equipment specifically designed as to size and weight for the particular child.

The last major point in our physical therapy program deals with teaching the child in a logical progressive manner the increasingly difficult physical accomplishments. This is an unremarked and unheeded basic phase of physical therapy and consists of establishing the level of physical development and then working up to the next level. This progressive training must be carefully supervised.

At any level of performance, multiple avenues of approach must be employed to teach mastery of a specific activity. As an example, the teaching of sitting balance, seemingly quite simple, is actually complicated because of the different normal positions of sitting and the various physical acts required to maintain balance. A child must be taught to balance his head, sit erect, recover his sitting balance, keep his feet flat on the floor, and, in fact, push with his feet against the floor to maintain balance. These inherent reflex acts must be taught individually to the cerebral palsied.

The child is ready for standing practice only after he has achieved sitting balance. Standing is begun with support for the legs, as head and trunk balance have been mastered previously in the sitting practice. The support for the legs is gradually decreased as independent standing balance is established. Finally, the child is ready for the most Herculean task of all, walking, which is balance in motion. This is the final goal and there are no short cuts. Parents must be counseled repeatedly in this progressive functional plan in regards to its background, reasons for being, broad implications, and final aims. This is imperative if wholehearted parental cooperation is to be obtained and the program to succeed.

Occupational Therapy

The hand is a complex piece of machinery possessing a matchless degree of facility of motion and sensory perception. Any disability of this complex structure creates a problem which is difficult to overcome or decrease. Traditional therapy relegated upper extremity training to the occupational therapist, and the lower extremities to the physical therapist. This was wrong and led inevitably to segmental analysis of the patient rather than integration of the whole child. This misconception has now been corrected and all spheres of training are correlated to reinforce each other. These two departments are now integrated so that the function developed and learned in one department will be applied in other spheres of activity. We believe, for example, that as a child develops greater proficiency in dressing himself, he will achieve greater skill in holding a crutch for walking. In a similar fashion, sitting balance taught in physical therapy will permit a higher degree of proficiency at a typewriter in occupational therapy.

Functional training in occupational therapy must be the paramount consideration. The ability to fingerpaint, draw a circle within a circle, or pile blocks is of value only if it aids in practicing reach and grasp. The neuro-muscular patterns of reach and grasp and hand-eye coordination are part of normal development but in the cerebral palsied these patterns must be taught. Until these neuro-muscular patterns are mastered the teaching of more complicated or involved physical procedures is but wishful and wasteful training.

The sphere of influence of occupational therapy should extend into the home through supervision of the child's daily activities of feeding, dressing, and self-care. Parents desperately need guidance in this realm. The therapist recommends and helps to construct the specially designed toothbrushes and eating utensils with special handles or grips. The clothing is checked to see that the buttons or hooks are readily accessible and that zippers are used wherever possible. Too often little or no thought has been given these problems so that a child who has the potential of self-care has remained totally dependent. This is a double calamity—the parents are burdened with extra work and the child is deprived of independent action.

Each accomplishment that promotes more normal and useful living and meaningful employment of the hands is progress in the right direction. The belief arises all too often in the parent's mind, consciously or unconsciously, that all necessary treatment and practice is received in the regular occupational therapy training period. This is fallacious, for the maximum physical potential can be realized only if the parents maintain an "around the clock" program at home. This is quite evident in the improvement among children whose parents or guardian have a sincere interest and participate actively in the care of the child. This statement is of course no less true for a handicapped child than it would be for a normal child.

Bracing

Bracing has become a stigma of cerebral palsy. Close your eyes and imagine a cerebral palsied child and you see the image of a helpless individual encased in heavy braces. Bracing may be a critical requisite, but all too frequently it is generously overdone. The weight and stiffness of large braces effectively anchor the child and eliminate any possibility

of self-locomotion. Braces do furnish secure and convenient handholds for the parent or therapist while the child is receiving therapy. Immobilized in braces of this nature the child can be goose-stepped about the house like a wooden soldier. The child's reactions to this type of walking are (1) complete frustration evolving from the suspicion that wooden soldier walking will be permanent, or (2) the child may be mesmerized into the belief that this is walking and be quite happy.

Tragically this robot walking may be doggedly continued by the parents well into the child's maturity. Certainly if walking were to have been learned by practicing this method it would have been established before age 20. In adulthood this type of walking demands an all-out physical effort due to the size and strength of the child. We have witnessed parents who literally have broken their backs physically, and their minds psychiatrically. A trying doctor-parent relationship arises when the attempt is made to alter fixed notions regarding this "walking." The change in treatment that restores the child to his proper functional level and decreases the bracing to a functional level may appear to the parent to be a backward step. The child must, however, learn to perform within his physical capabilities. Parents must accept a change after years of goose-step marching towards the mirage of independent walking. The child cannot learn to walk in this fashion.

We have progressively decreased the extent of individual bracing in the past ten years. However, braces which promote and facilitate self-activity are used extensively. Braces which require additional help to make them function have been largely discarded. Occasionally full control braces are necessary for a severely handicapped child who cannot otherwise use the standing table. All children practice standing several times daily if this is feasible. This, regardless of the final prospects of walking, promotes better body physiology.

The Frejka pillow splint, which was originally developed to treat congenital dislocations of the hips, is used extensively to prevent scissoring of the legs. The prevention of scissoring will make crawling, standing, propelling a chair, sitting, and in some cases, walking, easier. The common equinovarus deformity of the feet is controlled by short leg braces which produce a stable, flat, weight-bearing surface. Elastic twisters may be employed on small children to overcome the internal rotation deformity of the legs. A twister attaches to the shoe near the fifth toe and spirals up the leg through one turn to anchor at the pelvic belt. This device exerts a constant external rotation force while the child is standing. Short forearm braces which cock the wrists and hands into functional position are the only braces of value in the upper extremity.

Surgery

The subject of reconstructive surgery in cerebral palsy has been the focal point of numerous polemics. Advocates of surgery optimistically employ reconstructive procedures, believing that any procedure is worthwhile that will possibly decrease the physical disability or improve the cosmetic appearance. Their patients run the gamut of orthopedic surgery with neurectomies, tenotomies, bony stabilizations, tendon transfers, and osteotomies.

Those among the conservative school, disillusioned and discouraged with surgical results, have placed almost exclusive emphasis on bracing, therapies, and education. They maintain that surgery is not an unmitigated blessing, and instances have been reported in which increased disability has been the patient's final surgical reward.

A neutral position exists for physicians who see elements of value in both viewpoints. Surgical procedures at times may be of extreme value. The orthopedist must adopt an optimistic outlook seasoned liberally with realism in regard to the anticipated

(Continued on page 504)

Nothnagel's Syndrome

As It Applies to Vasomotor Angina and Paroxysms of Hypertension

LEW W. PURINTON, M.D., ***Wichita***

As a result of observing two cases of paroxysmal pulmonary edema, chest pain, and systemic hypertension the term Nothnagel's Syndrome as defined by Paul D. White and Thomas Lewis was investigated. Nothnagel's 1867 paper entitled "Angina Pectoris Vasomotoria" is reviewed. His four cases complained of varied peripheral sensations, anxiousness and many disagreeable thoracic sensations. The wide symptomatology, the paucity of physical findings and the vague terminology used creates doubt regarding the existence of hypertension and even true angina in Nothnagel's cases during an attack. Comments by various authors on Nothnagel's four cases are presented and the problems of translation and semantics are discussed. The use of Nothnagel's name in referring to parasthesias and unilateral oculomotor paralysis is touched upon.

It is finally concluded that the term Nothangel's Syndrome when implying paroxysmal hypertension with or without angina pectoris was inadvisedly applied to a series of four ill-defined and confusing cases which troubled Nothnagel over ninety years ago.

Since the term Nothnagel's Syndrome will be discussed below, some preliminary remarks regarding Dr. Nothnagel are in order. Carl Wilhelm Hermann Nothnagel was born in Brandenburg in 1841 and died in 1905. He was a pupil of Traube and Rudolf Virchow in Berlin. From 1865 to 1868 he was Leyden's assistant at Königsberg and it was during this period of time the paper under discussion was written. He served as professor at Freiburg in 1872 and Jena in 1874. From 1882 to the time of his death in 1905 he was professor of medicine at Vienna. He was a gifted orator, an impressive lecturer, and a man of high ideals. A victim of angina pectoris, he stoically set down his own symptoms in writing just before his end. Dr. Nothnagel is remembered for his 24 volume encyclopedic *Handbook of Special Pathology and Therapeutics.* He first described universal anesthesia or absence of all sensations in the body in 1887. His favorite clinical themes were the diagnosis of cerebral diseases (in which he separated disease of the optic thalamus), and diseases of the intestines and peritoneum on which he wrote a classic monograph in 1898.

In standard texts and in the literature the term Nothnagel's Syndrome is rarely found. As referred to in Dorland's Dictionary, Jamison and Mortons Medical Bibliography, Kelley's Encyclopedia of Medical Sources, and Grinker's Neurology; Nothnagel's Syndrome implies a unilateral oculomotor paralysis with cerebellar ataxia and crossed paralysis.

To Paul D. White and Thomas Lewis, Nothnagel's Syndrome means general vasoconstriction with or without angina pectoris. This paper will be directed toward the latter interpretation. Other authors to be quoted later refer to Nothnagel's paper on vasomotor angina but do not use the term Syndrome. This communication was prompted by the observation of two cases characterized by paroxysmal attacks of acute pulmonary edema, chest pain, and associated hypertension. It was initially felt these patients might represent Nothnagel's Syndrome as referred to by Dr. White in his "Diseases of the Heart."

This paper's purpose then is to discuss and define the term Nothnagel's Syndrome as it applies to vasomotor angina and paroxysms of hypertension. Obviously the information to be relayed today has more historical and psychological implications than scientific importance.

In Paul D. White's fourth edition of "Diseases of the Heart" the following statement is made. "It is possible that vascular crises (coronary spasms) are at times a factor in the production of angina pectoris. When there is a general vasoconstricting storm producing paroxysmal hypertension with or without angina pectoris the condition has been called 'Nothnagel's Syndrome.' "

Nothnagel's Paper

In 1867 H. Nothnagel published an article entitled "Angina Pectoris Vasomotoria." He presented four cases; cases one, two, and four were males whose ages were 38, 39, and 46 respectively. Case three was a 63 year old woman. The principal symptoms com-

plained of were: a feeling of deadness, sleepiness or numbness of the hands and feet; stiffness of the extremities—especially of the hands and feet; coldness of the hands and feet; crawling sensations over the extremities; severe heart palpitations; and anxiousness. (See graph I.) The peripheral symptoms were usually noted to precede the central or thoracic symptoms. Less common complaints were dizziness in two patients, sweating in two patients, and paleness of the face of two. Case number two complained of clonic cramps in his extremities and a pressure in his neck. Case three complained of precordial discomfort, left arm pain, and epistaxis.

In graph II it is to be noted that the only finding characteristic in all four was paleness of the hands and feet at the onset of an attack. Case three was found to have a lowered pulse rate, whereas the other three had a normal or unobserved pulse. The radial pulse was described as "small" in case one and as "small in circumference" in case three. The "pulse wave" was observed to be lowered in height in cases one and three. In cases one, three, and four the attacks were almost always produced by exposure to cold. Case two generally developed attacks after drinking.

In Nothnagel's discussion he describes the symptom "Anxiety" more fully. It took many forms such as pressing, emptiness or chest fright, a feeling of nameless anxiety, and a feeling of expiring or dying. These symptoms were always located in the epigastrium or the precordial area. Nothnagel also states that frequently a numb sensation was noted in the heart region and that in one case it spread all over the left chest and at times into the left arm. Although describing only four cases he leaves one with the impression of having seen many more. At no time does he use the term "Pain" in describing the symptoms associated with anxiousness. These symptoms might well have represented angina as we know it today.

SUBJECTIVE SYMPTOMS COMPLAINED OF
GRAPH I

Case Number				
1	2	3	4	
X	X	X	X	Deadness, sleepiness or numbness of hands and feet
X		X	X	Stiffness of extremities
X	X	X	X	Coldness of the hands and feet
X	X	X	X	Crawling sensations on extremities
X	X	X		Palpitations
	X	X	X	Anxiousness
X	X			Dizziness
	X	X		Sweating
		X	X	Paleness of face
	X			Clonic cramps
	X			Pressure in neck
		X		Precordial pain
		X		Left arm pain
		X		Epistaxis

PHYSICAL FINDINGS
GRAPH II

Case Number				
1	2	3	4	
X	X	X	X	Paleness of hands or feet at onset of attack
		X		Lowered pulse rate
X				Pulse normal
	X		X	Unobserved pulse
X				Arterial radialis "small"
		X		Arterial radialis "small in circumference"
X		X		Pulse wave lowered in height
X		X	X	Attacks produced by exposure to cold
	X			Attacks produced after bouts of drinking
X				Cyanosis of nail beds
X				Temperature in extremities lowered
X				Face pale
X				Objective loss of sensation to pin prick

Nothnagel felt his cases were similar to those of Heberden and that his own term "Angina pectoris vasomotoria" was preferable to Heberden's term "Angina pectoris" since it more fully described the pathology. Nothnagel felt the basic pathology was in the nervous tissue of the heart vessels and not in the arteries or myocardium themselves. He supports this theory with the following points: (1) the peripheral changes precede anxiety and chest symptoms; (2) frequently early in the disease only peripheral symptoms were present; (3) patients feel better on walking around during an attack; and (4) relief is obtained by warm applications, avoidance of cold exposure, and medication directed toward vasodilatation. He also felt that peripheral vasoconstriction existed and that this in turn produced an increase in peripheral resistance with resultant increase in heart action and palpitation. Anxiety and pressure he felt to be secondary to palpitations.

Controversy

A review of subsequent literature referring to Nothnagel's paper was made. In 1902 Gibson discusses organic and inorganic causes of angina and under the inorganic group he lists neurotic causes which he further divides into reflex agencies, vasomotor instability, neuroasthenia, and hysteria. He felt that in every instance in the neurotic group there would eventually be found co-existing cardiac weakness. He stated that cases resembling Nothnagel's

1867 description were exceedingly rare. Also, in 1902 Sir James Mackenzie stated that, "in vasomotor angina the symptoms indicated a period lasting usually some hours, during which the patient was cold and chilly, with lividity of the extremities, and this symptom was attributed to a spasm of the arterioles, such as occurs in Raynaud's phenomenon. After a time this arterial spasm was followed by an attack of angina pectoris. On this evidence the vasomotor group of cases of angina pectoris was supposed to be established." Mackenzie felt that vasomotor angina existed in patients demonstrating paroxysmal hypertension but felt the symptoms in these cases were not similar to the symptoms in Nothnagel's cases. Later Mackenzie states that, "Many of the so-called cases of vasomotor angina pectoris bear no relation whatever to those cases described by Nothnagel, and the term is now evidently as vaguely applied as before Nothnagel's description."

Pal in 1905 felt there were two causes for angina pectoris. The first was organic and consistent with our thinking of today. The other cause he felt to be due to a neuralgia of the sympathetics produced over the cardiac plexus without pathology of the coronary blood vessels. His interpretation of Nothnagel's cases was that of peripheral spasm in association with coronary artery spasm. He felt that true angina pectoris was a cramp situation of coronary blood vessels together with ischemia of the heart muscle—the so-

Blood Pressure

The rarity of Nothnagel's syndrome was alluded to by Mackenzie in 1910. He stated that the term vasomotor angina conveyed to many that the blood pressure was elevated. His own experience failed to confirm this impression in those cases with cold and chilly extremities felt to resemble Nothnagel's patients. He states, "I have seen and carefully studied a few cases of Nothnagel's vasomotor angina pectoris and can positively state that they come under a different category from the more common forms of angina pectoris due to disease of the coronary arteries." Regarding angina and hypertension Levine and Ernstene in 1933 observed 23 cases of typical angina pectoris and observed a definite and significant elevation of blood pressure during an attack.

In 1918 Hirschfelder mentions J. Latham who reported a number of cases which in spite of the occurrence of intense anginal symptoms did not run the usual course ending in sudden death and to these he gave the name "pseudo angina." Hirschfelder stated that Nothnagel threw much more light on the subject and quotes Nothnagel as saying, "We must interpret this symptom complex to indicate we are not dealing with a disease which arises primarily in the heart, but that the symptoms of sternocardia are of secondary origin and are brought on by a general spasm of the arteries."

Lewis in 1932 presented four cases of angina pectoris in association with aortic insufficiency and discussed Nothnagel's four cases. He stated that the term "Angina Pectoris Vasomotoria" was not too well chosen, that Heberden used the term "angina" to signify a sense of strangulation with anxiety and that Nothnagel appeared to have used it in a similar sense. Lewis felt that Nothnagel's use of the term led to much confusion of ideas and distinct maladies. He felt that Nothnagel's cases probably had blood pressure elevation because of the pallor of the hands and feet although he admits this is inconclusive evidence. Lewis stated that facial pallor and sweating were not associated and thus he felt even more certain of hypertension being present.

It is to be noted, however, in graph I that cases two and three complained of sweating and graph II shows that in case one facial pallor was observed. Lewis suggested that Nothnagel's syndrome may be interpreted as constituting an exaggerated response on the part of the vasomotor system to cold exposure. In an earlier publication Lewis appropriately commented, "If we say that Nothnagel's syndrome is consistent with general vasoconstriction, and that this would explain the symptoms displayed by his patients, we probably state quite as much as is justified. It seems unreasonable that we should continue to base discussions of vasoconstriction upon what is largely guesswork while the essential facts are there for the collecting; or that we should continue to be saddled by terms which, by implying more than has been proved, tend to confuse our thoughts."

Mistakes Due to Semantics

It would thus seem apparent that confusion and differences of opinion exist regarding Nothnagel's four cases. In part this is a problem of semantics as evidenced by the use of such terms as "small arterial radialis," "reduction in circumference of the arterial radialis," "anxiety," "angina pectoris vasomotoria," and "pulse wave." It is entirely possible he meant something different from our interpretation today. His meaning of "anxiety" has been discussed above.

The presence or absence of hypertension during an attack (as described by Nothnagel in his article) is largely a matter of guesswork. The method of obtaining the pulse wave is not defined, however, the correlation between the pulse wave and the arterial blood pressure has long since been proven unreliable. It should be pointed out that Sigfried Karl Von Basch recorded the first blood pressure recording on humans in 1880 and that blood pressure recordings were not popularized until Riva-Rocci's improvement on the

mercury manometer in 1896. This was nearly thirty years after Nothnagel's paper. Lewis believed hypertension to be present on the basis of peripheral pallor and lack of facial pallor yet it is to be pointed out that in two cases facial pallor was subjectively complained of and in one case objectively observed. Dr. White stated in his fourth edition of "Diseases of the Heart," that "where there is a general vasoconstricting storm producing paroxysmal hypertension with or without angina pectoris the condition has been called Nothnagel Syndrome." Of interest is that White's reference is to an earlier article by Nothnagel in the same year and in the same journal. This article was entitled "Zue Lehre von den Vasomotorischen Neurosen" which translated means "For the Theory of the Vasomotoric Neurosis." On review of this article it becomes obvious that Nothnagel gives an almost classic description of what we now refer to as Raynaud's phenomenon, Raynaud having described this entity in 1862. With this in mind one can perhaps understand the similarity between Nothnagel's four cases and patients with Raynaud's phenomenon.

Final Status

With only four cases of rather wide symptomatology and ill defined physical findings to work with it seems unreasonable to retain the term Nothnagel's Syndrome which, as applied by a few authors over the years implies paroxysmal hypertension on a vasospastic basis with or without angina. It is likewise impossible to give a currently popular diagnosis to each of the four cases. Ninety years has seen tremendous medical progress and it is only fair to state that many maladies are better understood today than even 20 years ago. One could list several conditions which might explain any one or all of Nothnagel's four cases, however, that would be entirely beyond the scope of this paper.

I would like to express my appreciation for the invaluable help given to me by Kurt Reissmann, M.D., of the University of Kansas Medical Center, and Werner Langheim, M.D., Resident in Surgery at the Mayo Clinic. Without their help in translation and interpretations of the original articles, this paper would have been impossible.

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Cardiac Resuscitation

Part II–Plan of Treatment

ALFRED M. TOCKER, M.D.; VAN S. PARMLEY, M.D.; AGUSTIN ARBULU, M.D.; H. M. HOLLADAY, M.D.; *and* IRENE GLEICHMAN, R.N.; *Wichita*

Success in Cardiac Resuscitation Depends on Restoration of Flow of Oxygenated Blood to the Brain Within Three or Four Minutes (All Other Considerations Are of Secondary Importance)

It is most important to separate the resuscitation procedure into two distinct steps:

1. The immediate emergency re-establishment of cardiac and cerebral oxygenation and
2. A more deliberate restoration of the spontaneous heartbeat.

Treatment of cardiac arrest should never be attempted unless at least two persons are present—one to attend to the respiratory problems and the other to massage the heart. One person cannot successfully carry out this treatment. Furthermore, resuscitation should seldom if ever be attempted outside of the operating room or emergency room.

Delay in initiating treatment, more than any other factor, accounts for failure to resuscitate these patients. Treatment begins when the diagnosis is suspected rather than proven. When the anesthetist sounds the alarm the diagnosis is not to be questioned. The surgeon and anesthetist should spontaneously and automatically move to initiate preconceived resuscitation measures.

One must overcome the natural hesitancy to accept without question and more careful checking this almost always unexpected and dreaded complication—especially during the more accepted and routine low-mortality operative procedures or during minor surgical procedures, to say nothing of the diagnosis following merely the induction of anesthesia before any incision has been made (See Table II, Cases 9, 17 and 23). The formidable treatment of open thoracotomy to expose the heart must be an automatic reaction when the diagnosis is suspected. Time is the factor of utmost importance. The heart will respond effectively to relatively light stimulation early, whereas even vigorous stimulation later will fail.

From the Departments of Surgery of the Veterans Administration, Wesley, St. Francis, and St. Joseph Hospitals and the Department of Anesthesia of Wesley Hospital, Wichita, Kansas. This study was supported by grants from the Leo McGuire Research Fund and The Sedgwick County Chapter of the Kansas Division of The American Heart Association.

First Step in Resuscitation—

CEREBRAL OXYGENATION

Success in cardiac resuscitation depends on the restoration of the flow of oxygenated blood to the brain within three to four minutes. All other conditions are of secondary importance. This is accomplished by artificially maintaining the circulation while the lungs are being artificially ventilated with 100 per cent oxygen. This is the sum and substance of the treatment of cardiac arrest. These two procedures can keep the blood oxygenation at a level sufficient to maintain life.

Cardiac arrest is the most common cause of death in the operating room. The death toll from this cause throughout the country and in our own state each year is a significant one. The great majority of these deaths are preventable. In the authors' opinion, physicians and hospitals have definite medicolegal responsibilities in cases of cardiac arrest—the former to attempt resuscitation and the latter to have available necessary apparatus, drugs, and instruments. The set-up should be checked frequently to make certain all is in readiness. The technic of cardiac resuscitation should be reviewed by surgical staffs at least once annually.

The surgeon requires but one instrument—a scalpel. All routine precautions are disregarded. It is not necessary to wear gloves. If the patient is wearing clothing such as an undershirt, incision is made through the cloth. Infection, comparatively speaking, is a minor consideration to be dealt with later. However, if the surgeon is working in a grossly contaminated field, he may pull his gloves off quickly before entering the chest (See Table II, Case 18). There is no need to be concerned about hemostasis. In fact, if on opening the chest one finds active bleeding and spurting arteries, he may well be thankful that cardiac action does exist, rather than chagrined by a

mistaken diagnosis. The incision is made in the left chest below the nipple from the sternum to the mid-axillary region through an intercostal space.

The various means and methods of massage should be mastered on an experimental animal. We have organized, with the aid of the local chapter of the American Heart Association, a practice experimental demonstration set-up which permits observation of the effectiveness of the massage by the surgeon. The best method for massaging the heart depends upon the size of the heart and the size of the surgeon's hands. One hand, the right or left, inserted as soon as the exposure permits, usually suffices. When the right hand is used, the thumb is usually placed anteriorly over the right ventricle and the other fingers over the left ventricle. When the left hand is used, the apex of the heart fits into the palm of the hand. Use of both hands may occasionally be more efficient —one in front of the heart and the other behind it. Or the hand may press the heart against the sternum. In small infants, pressure on the heart between the thumb and index fingers may be effective. The inability to compress the heart properly is second only to a delay in its compression as a major factor in the failures of cardiac resuscitation. An important feature is relaxation of the massaging hand—which must be an active process—performed quickly and precisely—to permit full dilatation of the heart for cardiac filling. Similarly, the systolic phase of massage must be deliberate—not slow and drawn out.

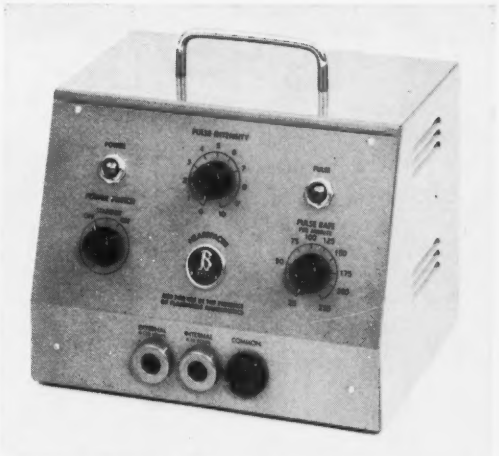
It is possible to rupture the heart, especially the upper and anterior right ventricular myocardium, by too vigorous manipulation[6] (See Table II, Cases 9, 10 and 17). This occurred six times in Wichita during a period of two years, including cases treated outside the operating room. Care must be taken to avoid pressure with the tips of the fingers. If the finger punctures the myocardium, the opening should be sutured quickly and massage continued. Successful resuscitation after such a perforation has been reported by H. Sloan, M.D., in Dr. Robert Hosler's "Manual on Cardiac Resuscitation" (Also see Table II, Case 17).

FIGURE 1

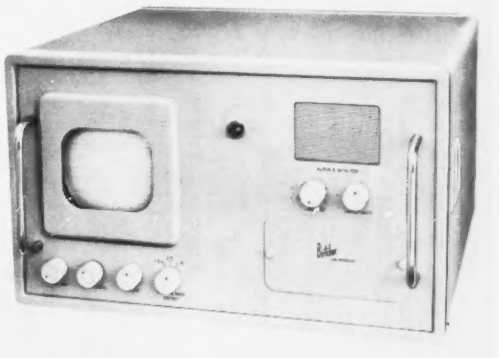
SPECIAL APPARATUS*

FOR CARDIAC RESUSCITATION

A. INTERNAL DEFIBRILLATOR. Ventricular defibrillator a *required* instrument in every cardiac resuscitation set-up. Special electrodes are available which assure against shock of the operator or burn of the heart.



B. CARDIAC PACEMAKER. This instrument is rapidly gaining popularity, particularly for use in the immediate post-operative period following cardiac resuscitation.



C. CARDIAC MONITOR. Attached to the patient during operation, this instrument aids in early and immediate diagnosis of cardiac arrest. It has gained considerable popularity for use in the immediate post-operative period following cardiac resuscitation.

* Courtesy the Birtcher Corporation, Los Angeles, California.

TABLE II

CARDIAC ARREST CASES
OCCURRING IN OPERATING ROOMS
WICHITA, KANSAS
1955-1958

1955

Patient	*Age*	*Sex*	*Procedure During Which Cardiac Arrest Occurred*	*Surgeon*	*Anesthetist*	*Died in O. R.*	*Recovered From Cardiac Arrest In O. R.*	*Sequelae and Remarks*
1	3	F	Division of patent ductus arteriosus.	1	A		Yes	No sequelae. Full recovery.
2	48	F	Exploratory lap. for chr. cholecystitis.	2	B		Yes	No sequelae. Full recovery.
3	66	M	Insertion of endotracheal tube following anesthetic induction. No operation performed. (Epidermoid CA vocal cord.)	3	A		Yes	Sequela: weakness.
4	57	F	Exploratory lap. for ileo-colic fistula (Inop. CA of colon).	4	B		Yes	Left hospital alive. No sequelae from cardiac arrest.
5	68	M	Inguinal herniorrhaphy.	5	A		Yes	Recovered from cardiac arrest but died 13 days later from acute internal hemorrhage (peptic ulcer). Was given cortisone.
6	48	F	Insertion of Hufnagel Valve for aortic insufficiency.	6	C		Yes	Recovered from cardiac arrest but died suddenly next day from acute internal hemorrhage (Hufnagel Valve slipped out of place).
7	20	F	Closure of patent ductus arteriosus. Cardiac arrest followed massive hemorrhage from aorta.	7	B		Yes	Died day after operation from effects of massive hemorrhage, multiple transfusions and surgically produced coarctation of aorta resulting from repair of opening in aorta.
8	45	F	Hemilaminectomy for ruptured cervical intervertebral disc.	8	C	Yes		Air embolism suspected as cause of cardiac arrest.

TABLE II—Continued

1956

Patient	*Age*	*Sex*	*Procedure During Which Cardiac Arrest Occurred*	*Surgeon*	*Anesthetist*	*Died in O. R.*	*Recovered From Cardiac Arrest In O. R.*	*Sequelae and Remarks*
9	42	M	Case of aortic stenosis (However cardiac arrest occurred during anesthetic induction.)	9	A	Yes		Cardiac arrest occurred during induction of anesthetic. Cardiac massage was undertaken immediately and carried on for one hour before resuscitation was accomplished. The right ventricle was ruptured during massage and successfully sutured.
10	61	M	Cardiac arrest in recovery room after partial decortication of right lung and drainage of pleural space.	9	C		Yes	12 days previously had had a right lower lobectomy for bronchiectasis. Oxygen was given immediately after diagnosis of cardiac arrest. The anesthetist massaged the heart within 3 minutes from the time arrest was discovered. After 25 minutes of massage the right atrium was ruptured. Died in recovery room.
11	60	M	Right pneumonectomy.	1	F		Yes	Full recovery. May be slightly euphoric.
12	3	M	T. and A.	10	A		Yes	Expired after seven hours.
13	17	M	Right pneumonectomy. (Bronchial adenoma in right main stem bronchus).	1	D	Yes		Death due to suffocation. A blood clot obstructed the left main stem bronchus after right pneumonectomy. While massaging the heart, clot was removed with a bronchoscope but it was too late for recovery.
14	5	M	Closure posterior palate.	11	A	Yes		Heart beat was restored and chest closed. Cardiac arrest recurred and chest was immediately re-opened. Second attempt was unsuccessful.
15	27	F	Emergency hysterectomy.	12	A	Yes		During the delivery of a 13 pound stillborn the uterus ruptured. Patient had massive bleeding and shock.
16	3	F	Ligation of patient ductus arteriosus.	1	A		Yes	Prior to cardiac arrest, pulmonary edema occurred in O. R. Patient died 4 hours after recovery due to respiratory failure.

TABLE II—Continued
1957

Patient	*Age*	*Sex*	*Procedure During Which Cardiac Arrest Occurred*	*Surgeon*	*Anesthetist*	*Died in O. R.*	*Recovered From Cardiac Arrest In O. R.*	*Sequelae and Remarks*
17	66	F	Intestinal obstruction due to incarcerated hernia.	13	E		Yes	Cardiac arrest occurred during induction of anesthetic. Heart perforated during massage. Perforation closed and resuscitation accomplished. After closure of chest, the obstruction was released and hernia repaired. Patient died 8 hours later in recovery room.
18	64	M	Transcolonic polypectomy.	6	C		Yes	Subsequent empyema (treated by chest drainage with recovery). Probably resulted from contamination from transcolonic polypectomy. No C.N.S. sequelae.
19	4	M	Pulmonic commissurotomy	1	C		Yes	The anesthesia was under hypothermia. During procedure, massive bleeding resulted from a tear of inferior vena cava prior to the cardiac arrest. Cardiac resuscitation performed. Patient died 12 hours later.
20	43	F	Exploratory laparotomy. Lysis of adhesions.	14	A	Yes		Peritonitis and obstruction followed hysterectomy. During the operative procedure, cardiac arrest occurred.
21	30	F	Mitral commissurotomy.	1	A		Yes	Died shortly after cardiac resuscitation.
22	37	M	Tracheotomy.	6	B		Yes	A gunshot wound of neck. Obstructed airways. Patient died 4 hours after cardiac resuscitation.
23	77	M	Internal fixation, fracture left hip.	15	C		Yes	Cardiac arrest occurred during anesthetic induction and was detected within 2 minutes. Resuscitated successfully after 4 minutes of massage. The internal fixation was thereafter accomplished. No sequelae.
24	87	F	Internal fixation, fracture left hip.	15	C	Yes		No massage.
25	29	M	Surgical treatment of abdominal gunshot wound.	2	B	Yes		The gunshot lacerated the superior and inferior vena cavae. Patient undoubtedly died due to massive blood loss.
26	50	M	Ventral herniorrhaphy.	16	B		Yes	No sequelae.

TABLE II—Continued

1958

Patient	*Age*	*Sex*	*Procedure During Which Cardiac Arrest Occurred*	*Surgeon*	*Anesthetist*	*Died in O. R.*	*Recovered From Cardiac Arrest In O. R.*	*Sequelae and Remarks*
27	53	M	Appendectomy.	17	A	Yes		During surgery the appendix was found to be perforated with localized peritonitis.
28	43	M	Aortic commissurotomy.	1	A		Yes	Ventricular fibrillation. Defibrillation delayed until defective defibrillator fuse could be replaced. Meantime cardiac massage was continued. Successful defibrillation and resuscitation.
29	3	M	Right inguinal herniorrhaphy.	5	E		Yes	Cardiac resuscitation was satisfactory. No sequelae.
30	65	M	Left pneumonectomy.	1	D		Yes	Cardiac resuscitation was satisfactory with no sequelae. Patient died 7 months after cardiac arrest due to extensive metastasis of bronchogenic cancer.
31	6	M	Craniotomy.	18	B	Yes		Craniotomy was carried out to evacuate a subdural hematoma. No response to cardiac massage.
32	4	F	Surgical correction of syndactyly middle and ring fingers, left hand.	11	B		Yes	Died in recovery room after cardiac rhythm had been restored by massage in O. R.

In shock, one of the quickest ways to give a transfusion is to elevate the lower extremities at right angles, thus utilizing gravity to introduce 500 to 800 ccs. of blood into the returning venous system.

Second Step in Resuscitation—

RESTORATION OF SPONTANEOUS HEARTBEAT

The immediate emergency re-establishment of cardiac and cerebral oxygenation, the first step in cardiac resuscitation, is accomplished by cardiac massage. Injection of drugs, if necessary, falls into the second step—the more deliberate restoration of the spontaneous heartbeat. The drugs recommended are discussed under "Cardiac Resuscitation Set-up." Again we wish to emphasize the importance of having a card setting forth the recommended dosages. The site of injection is of little importance, but the anteriorly-placed right ventricle is preferred because it does not bleed as easily as the atrium, it is more accessible and has fewer coronary vessels on its surface than the left ventricle, its wall is less thick than the left ventricle, and it does not have the large papillary muscles of the left chamber.

Electro-shock apparatus must be available for treatment of ventricular fibrillation (See Table II, Case 28), which occurs in about 10 per cent or more of the cases of cardiac arrest. While, under the worst of circumstances, the exposed wires of an electric cord wrapped in saline-soaked sponges may be used (in which case the operator should wear two pairs of rubber gloves), there is little excuse for failure to have available a ventricular defibrillator (See "Special Apparatus"). Imperceptible ventricular fibrillation may be revealed by an electrocardiogram, especially if the heart fails to resuscitate otherwise. The electrocardiogram should be disconnected at the time of shock.

We favor a high voltage of short duration (160 volts for 1/10 second in adult hearts). However, voltage may range from 60 to 160 volts, but 110 volts and 60-cycle alternating current is most practical because it is used almost universally in this country. A range of 1.5 to 3 amperes may be used, but 2.5 amperes applied for one second is satisfactory for most adult hearts. Voltage determinations differ among the defibrillators on the market, and the desired voltage for men, women, children and infants should be marked on each machine so that the operator, who may not be familiar with these determinations, may have some guide in this respect. Single shocks which fail to end the ventricular fibrillation are to be followed by a series of two or three rapidly repeated shocks. If this fails to accomplish the desired end, a series of six or eight short (1/3 second) shocks may be applied.

The heart musculature must be maintained in good condition by constant massage before and between the application of each shock. Care must be taken not to burn the heart. If special electrodes are not available to avoid such injury, saline-soaked 4" x 4" sponges may be placed between the electrodes and the heart musculature.

If there is any suspicion that massage is undertaken later than four minutes after the onset of the cardiac arrest, or if the heart fails to respond immediately, lowering the patient's temperature by the application of ice packs (particularly around the neck but also over the body generally) will help protect the brain from damage.

Treatment Outline

A suggested outline of treatment of cardiac arrest is set out below. A somewhat more detailed outline has been published by the Cardiac Resuscitation Committee of the Los Angeles County Heart Association. It is suggested that these outlines be posted in conspicuous places in the scrub rooms and included in the cardiac resuscitation set-up.

Each case must be individually evaluated before treatment is abandoned. Resuscitation attempts should be continued at least one hour after it appears that all hope is lost (See Table II, Case 9). Hosler reported complete recovery after eight hours of continuous cardiac massage.

After Care

Expert nursing constitutes the core of postoperative care. One must be constantly alert for cardiac arrhythmias and a recurrence of cardiac arrest. A cardiac

INSTRUCTIONS FOR TREATMENT OF CARDIAC ARREST

Respiratory:

1. Establish an airway.
2. Connect airway with oxygen supply.
3. Maintain artificial ventilation.
4. Lower head of table. Elevate legs.

Cardiac:

1. Enter left chest through an incision in interspace below nipple or breast extending from the sternal margin to the mid-axillary line.
2. Begin *massage of heart immediately.*
3. Improve exposure with rib spreader. If heart beat has not been restored at this point, open pericardial sac.
4. Massage the heart to produce a palpable peripheral pulse at the rate of 60 to 70 per minute.
5. Use cardiac emergency drugs as indicated.
6. In case of ventricular fibrillation, defibrillate. Then massage again.

(These instructions are to be carried out by the physicians present, be they interns, residents or members of the attending staff.)

monitor, with an alarm system, and a pacemaker, if available, should be attached. It is in this post-operative period that these instruments serve such useful functions. The monitor sets forth an alarm when threatening cardiac arrhythmias or cardiac arrest occur, and the nurse immediately flips on the pacemaker. Thus, precious seconds are saved, although doctors should be available to open the chest again if necessary. An emergency cardiac resuscitation set-up should be available at all times for this purpose, as well as a tracheotomy tray, to be used if the patient's tracheo-bronchial tree cannot be kept clear of obstructing secretions. Chest drainage should be used post-operatively to help assure full expansion of the lung. Elevation and wrapping of the limbs will aid in insuring an ample supply of blood to vital organs.

Antibiotics are used liberally. Levophed combats hypotension without accelerating the heart. Neo-synephrin is also a satisfactory vasopressor drug. Adrenal cortical extracts are useful as the adrenals often suffer from deprivation of oxygen. Other drugs (atropine, caffeine, citrate, aminophyllin, calcium gluconate, quinidine, Cedilanid®, etc.) are used as indicated. An electrocardiograph is necessary to diagnose cardiac disturbances.

Acute cerebral edema is usually the cause of death in those individuals whose circulatory systems have been restored to normalcy. Twenty five per cent human serum albumin in 100 cc. amounts may be given intravenously every four hours as long as it appears necessary. Fluids, including blood, must be administered judiciously. The room should be kept cool. Hypothermia may protect the brain at this stage. A rapid rise in temperature usually indicates cerebral injury.

Special Apparatus

"Be not the first by whom the new are tried,
Nor yet the last to lay the old aside."
From Pope's "Essay on Criticism"

Cardiac Monitors have not been used in most hospitals, generally, for every patient during operation. However, monitors are available which give visual and audible warning of actual or impending cardiac arrest. Some models automatically start the external pacemaker when the alarm stage is reached. The monitor has found use following operation in resuscitated cardiac arrest cases.

External and Internal Cardiac Defibrillators are available. The external defibrillator is useful only if electrocardiographic tracings are immediately available at the time of cardiac arrest revealing the existence of ventricular fibrillation. The internal defibrillator under the circumstances existing generally in Kansas hospitals at this time, is a definitely acceptable instrument for which there is no substitute. It is the one special instrument above all others which every hospital must have available.

External and Internal Cardiac Pacemakers are available. The *external* pacemaker is of questionable value. Medically it is being used more widely in the treatment of Adams-Stokes seizures.[11] An inlying intracardiac pacemaker, using cardiac catheterization technic to place and keep the stimulating electrode in the right ventricle for more than three months has been used successfully in the case of a 76-year-old patient suffering such seizures.[12] The *internal* pacemaker is used on the exposed heart to eliminate the trauma of manual cardiac massage. By means of electrically conductive sutures* placed in the heart, in cases in which resuscitation is unsatisfactory or in which a recurrence of the cardiac arrest is expected, a pacemaker can be attached for control of the heart beat. In many larger centers such sutures are placed in the heart routinely after cardiac arrest cases which are resuscitated with difficulty or do not show a good strong heart beat after resuscitation.

Mechanical Respirators, such as the Stephenson controlled respiration unit, take over the respiratory control in long and difficult resuscitative procedures.

Comments Regarding Special Apparatus

The occurrence of cardiac arrest, in our opinion, necessitates thoracotomy for cardiac massage, regardless of whether the heart is in a state of asystole or ventricular fibrillation. Reliance upon such apparatus as external cardiac defibrillators or external pacemakers at this stage will result in a higher mortality. The fibrillating heart must be massaged to improve the oxygenation of the cardiac musculature before it will respond to defibrillating electro-shock. Reliance upon external stimulating and shock apparatus is likely to cost precious seconds. The various alarm systems, too, are not completely reliable, and cannot replace the constant attention of the anesthetist to the patient's condition. The internal defibrillator is indispensable.

The cardiac monitors and pacemakers have their greatest value in the immediate post-resuscitation period.

Medicolegal Aspects

"It is a crime to let any patient with a good heart and a good pair of lungs die in the operating room without any attempt at resuscitation."—Claude Beck, M.D.

It is believed that the first malpractice suit for failure to perform cardiac resuscitation measures in a case of cardiac arrest was tried in Cleveland, Ohio

(Continued on page 512)

* A special cardiac electronic suture for this purpose and an adaptor for attachment to a cardiac pacemaker are manufactured by Ohio Chemical & Surgical Equipment Co. of Madison, Wisconsin.

Parenteral Feeding

A Study of Fat Preparations for Parenteral Infusion

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Since parenteral feeding has become a reality there has begun an earnest search for more usable preparations and better caloric materials. Fat in theory was a logical answer, as it has the greatest caloric density, exerts no osmotic pressure when in solution, and is not rapidly excreated by the body. However, the controversy over its utility as a parenteral emulsion was not easily solved. This study tells of many of the experiments, both animal and clinical, which helped answer this question.

History

The need for an energy dense parenteral fluid with a low osmotic effect has been realized by the medical profession for many years. Starting in 1679 there has been interest in experimental infusions of fat. However, it was many years before any real progress came in this field. As is usually the case in medicine, the answers came from many areas of learning and have been combined through the years into an increasingly clear understanding of the problem. In 1895, Leube suggested the oil vehicle which he used to inject camphor into cardiac patients might be of caloric utility for the body. In 1898, De Mesnil noted the protein sparing effect of fat in the diet. In the first decade of the twentieth century, numerous animal experiments with parenteral fat were performed. By 1918, emulsified fat had been given intravenously by Mansfield, but here again its administration was only as a solvent for camphor or quinine.[8]

The earliest attempts to provide patients with additional calories by intravenous fat were reported in Japan from 1920 to 1928 (Yamakawa and Nomura). These results were concurrent with animal success in this country. For example, several workers provided up to 25 per cent of the caloric requirements of rabbits with subcutaneous injections of lecithin oil emulsions. Holt, Tidwell, and Scott (1935) made the first attempt to provide intravenous calories by administration of fat emulsions in the United States. These tests were carried out in marasmic infants with evidence that the preparation was oxidized.[11]

Prompted not only by the need of a new nutritional supplement but also by the need for a method to administer a complete diet intravenously in order to study the production of essential metabolites by intestinal flora, much experimental work has been carried out in the past years. The results of this work have been encouraging, but have uncovered many problems which are yet to be completely resolved.

Utilization

One of the earliest studies of parenteral fat utilization was reported by Murlin and Riche (1915). These workers observed increased heat production and decreased respiratory quotient in dogs given emulsified lard. Baba, from 1931 through 1933, reported that his study of respiratory quotients of infused animals indicated the administered fat was burned for energy.[8] Gordon and Levine (1935) suggested evidence that intravenous preparations were oxidized in healthy infants. Utilization of infused fat emulsions was demonstrated by Narat (1937) in the extension of survival time when it was administered to starved dogs. By 1943 complete parenteral nutrition was accomplished in adult dogs receiving only water by mouth. In these studies, a preparation containing one calorie per ml. was infused along with other nutritional factors to provide about 40 calories per kilogram of body weight per day.[20] McKibbin and associates (1945) demonstrated the caloric utility of a preparation which produced no untoward effects during the injection periods. The laboratory reported that their fat emulsion prevented further weight loss and gave increased nitrogen retention. They also found that none of the infused fat was excreted in the urine or feces nor deposited to a special degree in any tissue. The character of the body fat remained unchanged. From these results, they concluded that the intravenous fat was metabolized.[19] The maintenance of nitrogen balance and prevention of weight loss for several weeks in adult dogs on caloric inadequate oral diets has since been repeatedly demonstrated.[5, 23, 24]

To approach the problem from a different aspect and to answer the arguments that previous experiments had measured factors other than true utilization, several groups of workers used radioactive

This paper was prepared while Dr. Olson was a Sophomore Student at the University of Kansas Medical Center.

tracers to follow the administered emulsions. Injected trilaurin in which the carboxyl group was labeled with C^{14} was studied in 1948. Seventy-one per cent of the C^{14} was recovered in expired air in four and one-half hours.[9] In another study, palmitic acid was synthesized with an atom of C^{14} at the sixth carbon atom:

$$C_{10}\ H_{21}\ C^{14}\ H_2\ CH_2\ CH_2\ CH_2\ CH_2\ COOH$$

This was then esterified with glycerol and an emulsion of the tripalmitin was made. In collecting the expired CO_2 and tabulating the C^{14}, 7 per cent was recovered in the first two hours and 50 per cent by 24 hours. Autopsy of the animals also showed that 50 per cent of the C^{14} found in the liver had been converted to phospholipids.[17] Deuterated fat was used in other experiments. Fifty per cent of the infused deuterium was recovered in the respiratory and urinary water within 24 hours. These experiments were more direct and provided unequivocal evidence of the oxidation of the parenteral administered fat.

Infusion into critically ill patients demonstrated utilization by negative nitrogen and potassium balance reversals even though the patients were on low or inadequate protein diets. In spite of the critical condition of these patients, no weight loss was noted during the time that fat calories provided an adequate diet.[28] Recently intravenous fat was shown to be as well utilized as oral fat emulsion. Both corrected negative nitrogen and potassium balances in healthy patients on subcaloric yet adequate protein diets.*[3]

Emulsions and Stabilizers

A stable infusion was necessary before any clinical or good animal experiments could be carried out. Certainly the untoward effects had to be conquered before fat emulsions could be utilized as a common nutritional supplement. It was speculated that a preparation which would resemble chyme in particle size (less than one micron) and dispersion would be the most satisfactory, as the body was already prepared to handle such material.

A variety of factors which are recognized to influence the behavior of colloids were considered in an attempt to produce the required dispersion of fat (an immiscible substance of low density), into an aqueous medium. These factors include the charge, concentration of the dispersed fat, specific gravity, particle size, and interfacial tension.[4] It is sufficient to point out here that stabilization of a fat emulsion requires the particles to be highly dispersed and stable in this state. There is a great tendency for such emulsion to "break" so that the preparation must be considered not only in its original form but also as it will be affected by heat, mixing with salts (equivalent to a .85 per cent sodium chloride solution), storage, and rough handling. Until recently it seemed improbable that any such an emulsion could be produced by simple mechanical means. Stability was made possible, therefore, by using an emulsifying agent. These were of two types: surface active wetting agents and hydrophobic materials. Specifically the stabilizer had to meet strict requirements. It had to be nontoxic, retain status quo through autoclaving and storage; the stabilizer-fat mixture had to be nontoxic and also economical and therefore commercially feasible. Considering the two types in general, the first (surface active wetting agents) are often toxic and cause hemolysis. The second type (hydrophobic colloids) seemed to provide the most success.

Particle Size

Particle size did not advance too difficult a problem as with a high pressure (3,000 to 4,000 pounds) homogenizer purified, relatively pyrogen-free material was possible at particle sizes near one micron. The stability of such preparations, however, was and is the cause of much difficulty. Purified soybean phosphatides were used in early work (along with a great host of others) and their clinical trials were fairly successful and are considered so today. With the advent of World War II, the work along these lines was greatly stimulated and a mass of animal and clinical tests heve been conducted since that time.

Lecithin and related phosphatides although most frequently used are acknowledged to be relatively unstable. Frost demonstrated that spontaneous hydrolysis occurred upon standing in solution. It was noted by Geyer and associates that a vasodepressor substance which was not present in freshly prepared phosphatides seemed to develop on standing in contact with air. Fractions with low depressor activity were either poor emulsifiers or affected by autoclaving. The stability of corn oil and coconut oil emulsions used by Mann, Geyer, Watkin and Stare in 1949, when evaluated as to their pyrogenic reaction formation, was not considered satisfactory. The mechanism of these reactions was not understood, but they appeared to be nonbacterial in origin. Brown, Ware, Slanker and Zinsser (1954) published their belief that nonionic surface active agents, such as "Pluronic F68," might be the answer to these problems of ill effects and chemical and physical stability. Samples of the fat emulsion were stable even when dextrose, normal saline, and gelatin were added. These samples were well tolerated except in a few cases of pain on injection, tremors, vomiting, and bleeding.[16]

Other agents that have been employed to emulsify fat for parenteral use include bile salts, polyglycerol ester of fatty acids, gelatin, fattyacid esters of polyhydric alcohols or sugars, cerebrosides, organic esters of phosphoric acid, substituted amines, and many

*Potassium balance was restored more than the nitrogen balance.

others.[8] In spite of all effort, no completely satisfactory fat emulsion has been produced with any of the above mentioned stabilizers. However, many have been found which give a low incidence of untoward reaction and have thus been used with some excellent results.

Some new ideas for the preparation of fat emulsions have been published in recent years. Payne, Lizzarine, Brot and Beal reported the preparation of a colloidal fat suspension in 1955. Fat was dissolved in absolute alcohol and added to pure water with agitation. This mixture was evaporated to recover the alcohol and reduce the volume to give the desired concentration. After filtering, the particle size was found to be less than 250 millimicrons. This preparation may be autoclaved as prepared or with a mixture of salt or dextrose. This sterilized product was reported stable to storage and satisfactory for infusion. No untoward effects were observed in any of the patients tested. This certainly would seem to be a great advance. The difficulties encountered in the use of a stabilizer are completely avoided. However, there have been no reports on its use since that time.

Shafiroff and Mulholland published a recent report concerning work they had carried out in the preparation of an "instant" fat emulsion. Clinical trials were not satisfactory, but the idea is sound and is being further explored. Such a preparation would eliminate the problem of stability during storage.

Untoward Effects

From the earliest attempts to the most recent reports the administration of fat has been plagued with untoward effects. The complete list of these effects would be far too protean to mention. The discussion will include only the more frequent changes. For the sake of both clarity and brevity several arbitrary classifications must be made. The effects may first be divided into two main groups, the first including those which have been solved, and the second those which are still uncontrolled. The second grouping may be further subdivided into four general types of response.

I. Resolved
  A. Aseptic peritonitis on intraperitoneal injection
  B. Pain on subcutaneous injection
  C. Cellular reaction
  D. Pulmonary embolism
II. Unresolved
  A. Pyrogenic reactions
    1. chills
    2. fever of less than four degrees Fahrenheit
  B. Blood pressure
    1. vasodepression
    2. flushing
  C. Blood cell changes
    1. lowered platelet count and prolonged clotting time
    2. decreased hemoglobin, hematocrit, and red blood cell counts
    3. increased cellular fragility
  D. Physical Discomfort
    1. nausea
    2. back and chest pain
    3. dyspnea
    4. headache
    5. blurring vision

The peritonitis and subcutaneous pain encountered in the use of fat emulsions in this first group are related in the most part to unsuitable preparation and unsatisfactory route of administration.[27] These have generally been avoided in more recent studies. The miliary cellular reaction of discrete, noncaseating necrotic, granulomatous appearance were seemingly either due to phagocytes becoming filled with fat or to a reaction to the foreign lipid. This was not believed to be inherent in fat emulsions *per se,* but specifically due to an unstable emulsion and the resulting embolism.[21] This was borne out by the same laboratory, and more satisfactory results with a new preparation were published the following year.[19] The host of early references to pulmonary embolism most probably can be explained by unstable emulsions.

Slight pyrogenic reactions often reported to be accompanied by a chill have been incriminated by most investigators as the most frequent untoward effect. Explanations for the pyrogenic effects of fat emulsions have been as numerous as the symptom itself. The reaction appears not to be caused by bacteria.[22] Neptune, Geyer, Saslaw and Stare pointed out that febrile state probably is not due to the rapid metabolism of the infused caloric rich material (as had been suggested by the other workers). They cited the results of a clinical study of nine seriously ill patients in whom no reactions of any kind were observed, either initially or after repeated feedings. These patients, in spite of obvious rapid utilization of the infusion, showed no elevation in temperature. It was suggested that the temperature regulating mechanisms of the body would not allow such a rise due to simple rapid metabolism. Even such rapid energy exchange states as thyrotoxicosis show only negligible changes in body temperature. Creditor related the blood changes he investigated (discussed below) to the febrile response. Feeling that fat infusions may be hemolytic in nature, he pointed out that diseases associated with acute intravascular hemolysis are characterized by the occurrence of fever. Waddell and associates considered both the emulsifier and a nonspecific colloidal phenomena to be in some part responsible for the fever. Thermogenesis was correlated with particle size by Brown and others,[16] and rabbit experiments by Becker and Grossman suggested pulmonary trapping of the fat was the basis of the rise in temperature. Fever, whatever the cause,

is in general not persistent and has produced no permanent effect.

Early Papers

Many early papers gave no mention of a vasodepressor response. This may be explained by the fact that soybean phosphatides do not have such an effect on rats or rabbits, and only a slight effect on dogs, while on man and cats the emulsifier shows active depression. The depressor substance is not found in freshly prepared emulsions but develops on standing. Shafiroff and Mulholland reported slight (10 mm. Hg.) elevation in blood pressure which was not correlated with particle size or sample age. All these reactions were short-lived and produced no after effects. Waddell, *et al.*, reported that the soya bean stabilizer caused a vasomotor change which elevated the systolic pressure while having no diastolic effect.

In studies of human patients Johnson, Freeman, and Meyer noted lowered platelet counts and prolonged bleeding time in association with intravenous feeding of fat emulsion. Creditor reported significant decreases in hemoglobin, red blood cell counts and hematocrit in a group of normal dogs after one to two weeks of daily intravenous infusions. He believed the explanation to be hemolytic anemia. This response could arise from the action of surface active emulsifiers, the fat itself might alter the interface between the erythrocyte and its environment, or free fatty acids in the infusion or liberated after infusion might become saponified and act as a surface wetting agent. In vitro, he demonstrated increased cellular fragility by subjecting the cells to mechanical stress. This test was carried out in the lipemic plasma, for if the cells are washed, no abnormality is seen. From this fact he concluded that the change in the red blood cell was not irreversible. As has already been mentioned, he suggested that there was direct correlation between the hemolytic and thermogenic properties of fat emulsions.

Physical discomfort has been reported with various emphasis and frequency and is most affected by standards of observation. Waddell, *et al.*, postulated visceral ischemia as the basis for back and chest pain, but no other specific explanations have been offered for other discomforts.

An excellent report of the typical findings of large scale clinical tests is found in a publication by Jordan, Wilson, and Stuart (1956). Excluding minor temperature side effects, the incidence of untoward reactions was 38 per cent and of these only six per cent were considered severe enough to stop the infusion. Back pain, flushing, chest pain, and dyspnea are usually apparent after only a few drops of the infusion had been given. All were transitory and stopped on discontinuing the preparation. In most cases the infusion could be started again with no ill effects. Chills and associated pyrexia did not occur before 100 to 300 ml. had been administered. If they did develop, a 15 to 20 minute rest period often was enough to allow continued infusion. Headache, nausea, vomiting, arthralgia, and blurring of vision were rarely observed before 500 ml. had been given. Seventy per cent of the cases in study were accompanied by elevation of temperature, but these were never really serious.

All of the untoward effects can be explained by one or a combination of four basic principles. Kinsell, *et al.*, suggested that an overload of the enzyme systems might well precipitate ill effects and advised a slowing of the rate of infusion. Waddell, Geyer, Olsen, Andrews, and Stare, working with fat emulsion, noted all of the reactions previously reported by Nissin after administration of colloidal iron directly into the blood stream. Jordan and associates postulated that nature of the colloidal reaction might be histamine in the early phases and an overloading of the reticulo-endothelial system in late reactions. Intravascular hemolysis must also be considered as an explanation for these untoward effects.[6] The reactions to repeated injections have not been well investigated in man, but the immediate reaction must be assumed to be due at least in part to a physiological factor within the patient. In no other way may response differences to various administrations of the same batch of emulsion in the same patient be explained.

Value in Man

The theory behind fat emulsion infusion is evident. Obviously a caloric deficiency which must be corrected by parenteral fluid therapy is of no particular problem in a reasonably well nourished patient for a limited period of time. It does become a problem in a patient with weight loss, who cannot or should not consume food by mouth.

The caloric containing materials usually available for parenteral use—glucose solutions, blood and its derivatives, and protein hydrolysates, do not provide enough calories in a reasonable volume. In order to raise the caloric level to a proper standard, the patient would drown in his own fluids. For example, a liter of five per cent glucose or protein hydrolysate yields only 200 calories. On top of this, the protein, if burned for energy, is of no value in body repair as a building block of tissue protein. Blood, for all practical purpose, has no immediate caloric utility. Dilute solutions of alcohol, invert sugars, and fructose have been tried in the search for a better caloric yield and although useful, have not answered the problem. Of all of these, alcohol has the greatest caloric density (seven calories per gram) and is a sedative and an analgesic, but there is evidence it may not be wholly metabolized. It cannot be given to

patients in shock or with demonstrated liver damage.

All of the above mentioned materials, except blood, are excreted in the urine or feces. By slowing the rate of infusion this may be decreased, but in so doing, additional strain is placed on both patient and attending staff. About 1,000 calories can be administered daily by these methods. Basal metabolic requirements of the average healthy adult is about 1,500 calories and this need often increases in disease. It, therefore, follows that fat with a high caloric density is logically an answer to the problem.* Another obvious advantage is that fats do not exert osmotic pressure and may be given in high concentration without the danger of thrombosis of the veins. For example, a 15 per cent fat emulsion with five per cent glucose gives an isotonic mixture containing 1,600 calories per liter. This compares with a 10 per cent glucose solution which besides being hypertonic gives only 400 calories per liter.

Fifteen Per Cent Emulsion

Such a 15 per cent fat emulsion was shown to be well tolerated in nine patients of the seriously ill, depleted category. No untoward reactions of any kind were observed and the patients lost little or no weight during its admission.[24] In 1952 Van Itallii, Waddell, Geyer, and Stare published an account of fat emulsion therapy in one case of complete intestinal obstruction. This patient was fed for 67 days entirely by the intravenous route until he was able to take oral nourishment. He was given 82,000 calories of which 42,000 calories were derived from 36 injections of fat emulsion. It was their belief that this patient would not have survived the illness without the additional caloric intake. They found no damage to the liver or erythrocytes, and concluded from their own, and other reports, that clinical administration of fat by intravenous method was an actuality. Others like Levenson, Lounds, and Morris agreed and helped stress the fact with reports of effective use of fat emulsions to provide high caloric intakes for burn patients. Kolff pointed out that protein feeding or inadequate caloric intake was deleterious in uremic states and suggested fat was probably more useful than carbohydrates. Parenteral feeding with intravenous fat emulsion was recommended by Van Itallii, *et al.,* in surgical patients in the later phases of convalescence where adequate caloric provision is essential.

Clinical Use

Concentrations of from 10 per cent to 30 per cent fat emulsions plus 4.3 per cent glucose to make isotonic solutions provide 1,200 calories to 3,200 calories per liter. Amounts up to five grams per kilogram of body weight have been given to adults and up to six grams per kilogram of body weight to a seven week old infant.[33] Fifteen per cent emulsion was completely satisfactory in nine critically ill patients.[28] It is important that if protein is to be given by the same method the best results are produced when the two are given at the same time.[1]

According to the findings of Kinsell, *et al.* 20 grams of fat per hour for six hours exceeds the tolerance of the average adult, but 10 grams for the same period and at the same rate was easily tolerated. Too rapid infusion apparently "overloads" the fat catabolic and storage enzyme systems.

Fat diets have been shown to decrease urinary volume and might, therefore, be contraindicated in some renal or cardiac diseases.[34] Patients with hepatic dysfunctions or adrenal insufficiency do not tolerate infusions as well as others, and if the emulsions are to be given it should be administered with added caution. Immediate postoperative patients also do not tolerate the fat as well, possibly due to altered hepatic function caused by the general anesthesia or because of adrenal depletion from the operative stress.[13] It has also been shown that although daily caloric intake is considerably increased immediately after the operation, there is no apparent change in the patient's progress for the first few days.[34]

Benadryl® (20 miligrams intravenously) is reported to stop back and chest pain and dramatically end dyspnea in many patients when the symptoms are caused by fat emulsions. Aspirin or Empirin® is generally sufficient to combat headaches. Dizziness can be prevented by keeping the patient in bed for 30 to 60 minutes after the infusion has been completed.[13]

The possibilities in therapy of burn patients and uremic states has previously been mentioned. Fat emulsions also hold considerable promise as the vehicle for intravenous administration of fat soluble drugs, especially vitamins. Here not so great a volume is injected and thus there is not so high an incidence of untoward effects. As an example, vitamin K preparations have been shown to be a strong antidicoumarol agent. Studies in man demonstrated that a prolonged prothrombin time due to dicoumarol may be reduced to the safe range within ninety minutes.[14]

Discussion

Definite proof of body utilization of the infused preparations has been given by radioactive fat studies. The definite proof of human utility, however, came some years later. Many workers, convinced that a proper emulsion would be of use in parenteral feeding, began to solve the problems of stability and compatability. This they accomplished by using various wetting and hydrophobic agents with high pressure homogenizers. No completely satisfactory prep-

* Fat has nine calories per gram, protein and carbohydrate have four calories per gram, and alcohol has seven calories per gram.

aration has been produced with the use of a stabilizer, but recent incomplete reports of a physical process have been encouraging. In the latter procedure, alcohol is used as a solvent for fat, then mixed with water, and finally removed by evaporation. This achieves so small a particle size (250 millimicrons) that the preparation has great stability.

Untoward effects have been a constant problem in fat emulsion administration. The reactions in general are very similar to symptoms seen on infusion of colloidal suspensions such as colloidal iron. The stabilizers used have also been incriminated as the cause of the ill effects. Both factors have been in some measure controlled, and it would seem there is hope that the reactions they produce may be completely eliminated by further study.

Concentration, infusion rate, and proper mixtures with sugar and salt have been investigated. These clinical results, although not extensive, present facts upon which safe widespread administration may be based. Certain types of patients have been found to be especially sensitive to emulsions and immediate postoperative patients have been shown to receive little or no benefit. Fat preparations have also been used as excellent vehicles for fat soluble drugs.

It would seem that the administration of fat emulsions is a possibility with present knowledge and that commercial production of the best preparation will come in the near future. It is improbable that fat will replace dextrose as the most common infusion, but it may well become standard operating procedure for long term intravenous feeding.

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Active Treatment of Cerebral Palsy

(Continued from page 485)

final surgical results. A pessimistic attitude would condemn children capable of gratifying surgical improvement to their deformities and disabilities. This is a greater sin than experiencing an occasional poor surgical result.

A limited number of surgical procedures have proven effective in cerebral palsy. Joint fusion is a successful procedure for deformed, unstable feet, or for flexed, contracted wrists. Tendon transfers are doomed to ignominious failure if the transfer involves a change in muscle function or joint action. However, it may be helpful simply to shorten and transfer a tendon insertion so that one joint is bridged, as in the Egger's transplant at the knee. Neurectomies and tenotomies undertaken after contemplative muscle and joint analysis can result in improved function and can decrease the possibility of joint contractures. These procedures, although technically simple, are not innocuous. Achilles tendon lengthenings have converted "apparent" equinus foot deformities into the far more disabling calcaneous deformities. This catastrophe stems from failure to recognize that the feet are not in true equinus but are simply reflecting hip or knee flexion deformities. Correction of the primary problem will restore the feet to their proper weight-bearing relationship. Obturator neurectomies and adductor tenotomies are indicated in "hopeless" cases to facilitate personal hygiene and simplify nursing care.

Reconstructive procedures should be reserved for children proven capable of improvement on a training program. Ambulatory, self-sufficient children may be benefited by surgery for a specific functional or cosmetic goal. A normal mentality is a desirable, but not a rigid, surgical criteria. The final goal is to make these children as self-sufficient as possible.

Chemopallidectomy has been tried by other workers in cerebral palsy. The permanent ablation of basal ganglia function has potential application in severe tension athetosis. Chemopallidectomy is still an investigative procedure, for long-term case studies are as yet unavailable. Although excellent results have been reported in a few cases, there is a three per cent mortality rate and a 30 per cent clinical failure rate associated with this procedure.

Medicine

Each year, like the burgeoning flowers of spring, enthusiastic reports of new medicines with the most salubrious effects on the cerebral palsied appear in the medical journals. These medications have uniformly failed to stand the test of clinical reanalysis, and we have never been able to approximate reported improvement rates. All the cerebral palsied children at the Institute of Logopedics are kept under close medical supervision. This situation creates an ideal testing ground for drugs. The double-blind method for drug administration has been employed. The medications are administered by a physician who is not permitted to know which is the placebo and which is the active drug. Both drugs are then administered with equal caution, and the result is a fair clinical trial. Glutamic acid, carbon dioxide inhalation, Tolserol, Flexin, and Paraflexin have failed under these test conditions to exert a beneficial effect on cerebral palsy. We are of the unalterable opinion, however, that a muscle relaxing drug will be discovered. Therefore, with this sanguine outlook, we plan to continue testing each drug that offers a reasonable assurance of success.

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If we consider men and women generally . . . there is only one situation I can think of in which they almost pull themselves up by their bootstraps, making an effort to read better than they usually do. When they are in love and reading a love letter, they read for all they are worth. They read every word three ways; they read between the lines and in the margins. . . . They may even take the punctuation into account. Then, if never before or after, they read.

—*Mortimer J. Adler*

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Committee
REPORTS

Defective Hearing in Children

Part I–Lay Audiologist

C. W. ARMSTRONG, M.D., *Salina*, Editor

In the evolution of preventive medicine in our State, slow and progressive changes occur usually after consultation with physicians. This is true in the school programs to detect children who have hearing losses that interfere with their ability to hear and therefore to learn.

For the past several years, a new school program has been operating which is worthy of the attention of Kansas physicians. It is hoped that the program for detection of hearing losses will be well received by physicians because it represents an attempt to assist in a problem of steadily growing proportions. The school audiologist is an individual who represents a group of laymen who are interested in the hearing problems of the school population. The audiologists have varying degrees of training and ability. The quality of work done, therefore, extends over a range. The range varies from a rather crude screening of the hearing capacity of the child (consisting of how well he hears a 4,000 cycle tone) to a rather complete examination of the child's hearing-communication capacity in terms of not only audiometry but also an assessment of speech reception of the individual.

This range of talent is represented, in general, by four groups. First, there are teachers who are assigned to help perform physical examinations on school children annually and test with whisper and spoken word the child's ability to hear. Others of the same group are using a simple form of the audiometer. Second, there are school nurses under the direction of the local Boards of Education who have

A series of six articles on Defective Hearing in Children will be printed in the *Journal* in the next several issues. These articles were prepared by C. W. Armstrong M.D., Salina, Kansas, for the Committee on Conservation of Hearing and Speech. The committee members are V. R. Moorman, chairman, C. W. Armstrong, J. A. Budetti, R. A. Draemel, E. L. Gann, C. L. Gray, W. P. McKnight, E. E. Miller, Ruth Montgomery-Short, W. D. Pitman, R. R. Preston, G. O. Proud, R. E. Riederer, and M. J. Ryan.

The titles of the articles will be Lay Audiologist, Defective Hearing in the Preschool Child, Defective Hearing in the School Age Child, Interpretation of Audiograms, Evaluation of Clinical Data, and Conclusions.

This is the first article in a series of six prepared by the Kansas Medical Society Committee on Conservation of Hearing and Speech and edited by Dr. C. W. Armstrong, Salina. The next article will appear in the December issue of the JOURNAL.

been given basic training with the audiometer. Third, there are audiometric technicians trained by the Department of Special Education of the State of Kansas for public school work. Many physicians have come in contact with one or more persons of these groups as well as the fourth group which is more highly trained. It is composed of the speech therapist who is trained in both audiology and speech therapy. The first reaction to the initial contact with members of these groups has been one of surprise to local physicians and there will be more physicians surprised as members of this group make their more or less unexpected appearance in new localities.

These individuals comprise the beginning of a new professional subsection of medicine. They are willing workers who are dedicated to their duties of locating, screening, and assisting the child with poor hearing to a source of help. At the present time, an outline of ethical conduct is beginning to develop.

Following the original screening in hearing, the child whose loss appears to be severe enough to interfere with his or her education is referred to the family physician by the person making the hearing test. If something is known by the doctor concerning the background of the referring examiner, he will have a clearer picture of the situation. If he knows the work to have been of the simplest form, he will know that a rather complete survey will be needed to assess the patient's condition. If he knows the examination to have been only an audiogram, he will want to make a brief audiogram re-check for record purposes and proceed to other forms of testing and examination. However, if he is fortunate and knows that one of the men or women with extensive formal training has done the tests, he will be able to accept the records sent to him at face value and proceed as if the audiogram had been performed by himself or by a technician in his employ.

Gross tests for hearing such as the watch test, whispered voice, spoken voice, and tuning forks cannot be calibrated and have a low degree of retest reliability. At the present time when used alone, they are considered unsatisfactory for good clinical work. In the event that the family physician feels that the scope of hearing assessment is outside his normal zone of interest, he may with good ethical conduct refer the patient to an otologist or a fellow physician, who is interested in otology, to obtain audiometer tests and consultation as to recommended treatments.*

* For those who are interested in obtaining a low priced useful audiometer, it is suggested that the Ambco Otometer, manufactured by the Ambco Company, Los Angeles, California, and priced at approximately 150 dollars is approved for screening purposes by the Committee on Conservation of Hearing and Speech as of October 26, 1958. Other audiometers with which more complete testing can be done are available.

It is well known that there are localities where there are no certified otolaryngologists and the burden of a reasonable amount of otology must fall on the shoulders of those general practitioners who have equipped themselves to assume the burdens of this amount of otologic work.

The problem of evaluation of a child's hearing loss lends itself to either good or poor public relations for the physician to whom the child is first referred. If he is not prepared to adequately evaluate the hearing and make definitive recommendations, he should refer the problem to a qualified physician when he becomes aware of the reason for the consultation.

(To be continued in the next issue of the JOURNAL.)

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Maternal Mortality

The patient is a 24 year old gravida V para IV who died with certificate diagnosis of "uremia due to lower nephron nephrosis following a transfusion reaction which occurred after a Cesarean section for placenta abruptio." The general level of medical and hospital facility in the community was good. The infant in this pregnancy died about 20 minutes after delivery.

The patient's medical history was not significant except for the obstetrical episodes. Of the four previous pregnancies, one progressed to eight months, the others delivering at earlier stages, the present one terminating at 27 weeks. The first child was hospitalized for "kidney poisoning" for the first three months of life. The second died in the neonatal period and was thought to have been jaundiced. The third was jaundiced but survived. The last child was grossly erythroblastotic.

Prenatal care was initiated in the first trimester and was adequate both clinically and from the point of laboratory procedures. As had been the case in previous pregnancies, the patient's blood was found to AB positive.

At 27 weeks of pregnancy, the patient was admitted to the hospital because of spontaneous rupture of the membranes. She was kept at bed rest for three days at which time bloody discharge commenced. On the fourth day definite bleeding developed. By the end of the fourth day the physican felt that the amount of bleeding was sufficient to require the delivery of the patient and that she did not represent a likely candidate for induction, therefore should be delivered by section. The fetal heart was not positively heard during this day. The doctor's feeling on the matter was supported by informal consultation in the hospital but the actual performance of the Cesarean section was deferred for several hours because of the lack of availability of a surgeon. The patient's blood was crossmatched with that of several AB positive donors and although the donors' blood proved to be compatible in saline, none would match in albumin. During the section, dextran was administered and a pint of blood from the donor whose blood showed the least agglutination on crossmatch was given. The patient was operated in the evening and by the next morning was severely jaundiced and was oozing blood from the mucous membranes and incision. For the following week, the jaundice continued and the patient was managed by dehydration consisting of intravenous fluids in the amount of the previous day's output (100 to 300 cc approximately). The patient improved and was able to be out of bed and around her room by the 12th day at which time evidence of cerebral irritation appeared and, subsequently, the patient went into convulsions. A comatose condition with increasing fever ensued and she expired on the 17th postoperative day. A subsequent check of her blood indicated that the patient was actually Rh negative. The patient's blood and the typing technique was studied by the reagent company providing the material for typing and crossmatching in the hospital. The company recommended certain changes in the procedure and the materials in the effort to obtain more accurate reporting in the future. An autopsy was not obtained because of the feeling that the death was due to the metabolic disturbant from the lower nephron nephrosis.

Committee Opinion

The committee believed that this was an avoidable obstetrical death. The history should have indicated to the physician the likelihood of an Rh factor problem or a ABO problem which would have led to more complete and illuminating studies before delivery. It was the feeling of the committee that every obstetrical patient should have blood typing with each pregnancy. Certainly, any blood of questionable compatibility should not be given as a transfusion. The incidence of all kinds of transfusion reactions in this country is approximently 5 per cent and this possibility must be kept in mind in the giving of any transfusion.

Classification

Maternal death, obstetrical, avoidable.

One of a series of case reports prepared by the Committee on Maternal Welfare to illustrate the type of study made in each instance of maternal death in Kansas.

Student
THESIS

Quantitating Acute Pulmonary Hypertension in Dogs

THEODORE G. HOSTETLER, M.D., ***San Antonio, Texas***

In our work on the experimental production of chronic pulmonary hypertension in dogs by the repeated injection of carborundum particles intravenously over several weeks, it was observed that the rapid infusion of small amounts of this embolic material caused acute pulmonary hypertension with right ventricular dilatation and death. But, if a sublethal dose was given, it could be repeated over several weeks until many times the acute lethal dose had been given. An attempt has been made to quantitate the lethal dose in respect to time, size of the dog, and pulmonary pressure.

Review of the Literature

Since pulmonary embolism in man is quite frequently fatal and still the object of speculation, it has been the subject of much research. Qualitative studies have been done on animals to determine the effects of pulmonary emboli. Few papers were found listing the quantities of material necessary to produce symptoms or death. A wide variety of embolic material has been used by many people for many reasons. Table I lists the embolic materials which have been studied. These are divided into three groups. Group I are massive emboli capable of blocking the main pulmonary artery. Group II are intermediate in size and Group III are those that block the arterioles and pre-capillaries.

TABLE I

Embolic Substance
Group I (Massive Emboli)
Autogenous Blood Clot
Catheter With Balloon Tip
Venous Catheter
Penrose Catheter
Soft Paraffin
Air
Group II (Intermediate Size)
Pea Seeds
Poppy Seeds
Filter Paper Fibers
Group III (Arterioles and Pre-capillaries)
Starch Suspension
Glass Beads
Carborundum
Lycopodium Spores
Barium Sulfate Suspension
Fats
Human Amniotic Fluid

The lethal doses reported in acute experiments on dogs using various emboli are given in Table II. W. H. Knisley injected 0.20-0.25 gm/kg of 50-150 microns glass beads in rabbits and found this to be a lethal dose. N. A. Halasz and J. P. Marasco found a total dose of 2 cc. of liquid omental fat was fatal in most of their dogs in 15 minutes and all were dead within one hour. None of the investigators sited in Table I attempted to correlate the body weight of the

This is one of a group of theses written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group pledged to be the best by the faculty at the school. Dr. Hostetler is interning at Fresno General Hospital in Fresno, California.

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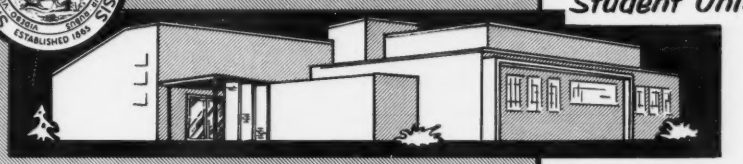

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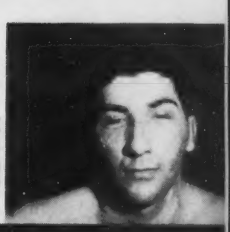

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animals with the pulmonary artery pressures or the lethal doses.

Mechanisms of death in pulmonary embolism are still speculative especially since there is the possibility that some emboli pass through the lungs via arteriovenous shunts to produce effects elsewhere in the body. Such pulmonary arteriovenous shunts have apparently been demonstrated in a few instances. There is by no means agreement as to what alterations in physiology occur as a result of emboli. A controversy has existed for decades over the presence or absences of reflex contraction of other vessels stimulated by particles locally, by the larger pulmonary arteries, the right auricle or ventricle, or the great veins. There are many supporters to the belief that such reflexes are not present, but that the mechanical factor of obstruction is sufficient to explain the sudden rise in pulmonary arterial pressure, the reduced systemic blood pressure and death. An equal number feel that some type of reflex mechanism is present that potentiates the effect of obstruction of the embolus by vascular spasm. A third and, admittedly, less likely possibility is the liberation of some toxin or humoral factor. Thus, the three possibilities for the acute hypertension following embolization are mechanical, reflex, and humoral.

TABLE II

Embolus	*Dose*	*Survival Time*
Starch	0.5 cc	2-5 min.
Barium Sulfate (sat'd sol.)	1.5-3.5 gm. (2 dogs took	———
Glass Beads (60-420μ)	5 gm.) 5-8 gm.	2 hours

Saddle Embolus

Experimental work indicates that a single embolus is fatal to an experimental animal only if it is a "saddle embolus" obstructing the main pulmonary artery. The clinical fact that a complete right or left pneumonectomy does not alter the pulmonary arterial pressure significantly is often cited. The point at issue is the effect of miliary emboli on the vital functions of the host. Perhaps miliary emboli have far-reaching effects from the following evidence. First, at autopsy small emboli are frequently found in the lungs. Second, it has been observed that papaverine seems to have beneficial effect in the treatment of pulmonary embolism. Third, in experimental animals the injection of barium sulfate suspensions into one lobar artery of rabbits and dogs produces a rise in pressures in the other lobes, although others have been unable to confirm this. The fact that small emboli are found in the lungs at autopsy is no indication that they were the cause of death. Similarly, the experimentally observed action of papaverine on smooth muscle is no indication that this drug relaxes pulmonary vessels in spasm since these vessels may have been of normal caliber and papaverine merely dilated them further. Dr. W. D. Holden has actually seen papaverine increase pulmonary arterial pressure thus contributing to the load of the already overworked right ventricle. Similar effects of papaverine have been observed in our laboratory. A. H. Niden and D. M. Aviado observed the effects of size of glass beads on the pulmonary arterial pressure in dogs. If glass beads greater than 200 microns in diameter were used, they saw an initial increase in pulmonary artery pressure followed by a leveling off and stabilizing at that level. In glass beads less than 160 microns in diameter, two responses were seen. First, there was a prompt rise in pulmonary artery pressure then seconds later was another gradual rise of less magnitude. They postulate the smaller beads penetrate deeper into the pulmonary vasculature and obstruct smaller vessels. At this site is the origin of reflex activity which, they believe, accounts for the second rise. They explain the initial rise on mechanical blockage of the pulmonary vessels. I have observed as has W. H. Knisley that small emboli tend to clump together as they enter the blood stream and even single particles have been seen to be coated with a white precipitate immediately after entering the blood stream. This makes it more difficult to be certain minute emboli stay small and do not become larger by clumping.

Another objection to the use of miliary emboli is the possibility of the particles passing through the lungs and causing additional effects outside the lungs. As mentioned above, arteriovenous shunts may be large enough to allow small particles to pass through. Suspensions of starch and barium sulfate have been demonstrated in the abdominal aorta of rabbits after embolization.

Pressure Changes Signals

If reflexes are present, what are their pathways? D. Whitteridge describes at least one type of fiber that appears to signal pressure changes in the pulmonary arterioles or capillaries. He believes these are the fibers that are concerned with pulmonary embolism although he offers no definite proof. J. J. Byrne studied vessels under 20 microns in diameter in the lungs of rabbits. He used barium sulfate with particles less than five microns in diameter. If there was no vasoconstriction the particles would pass through the lungs and lodge in the rest of the body. When vasospasm occurred, the particles were trapped

in the arterioles. The control animals after embolization had 20.5 arterioles per low-power-field (LPF) filled with emboli. After tetraethylammonium and tolazine only 6.9 and 3.7 arterioles, respectively, had emboli. His conclusion was that using five micron emboli in rabbits there was sympathetic activity associated with it which can be blocked by tetraethylammonium and tolazine. Others would agree with this, but the dissenters are numerous. V. V. Parin in his work on open chested cats found a "depressor reflex" in systemic circulation mediated by the vagus. The work of G. E. Hall and A. H. Ettinger does not seem to substantiate this.

A physiological vasodilator of the pulmonary bed is oxygen. Conversely oxygen lack results in vasoconstriction although no evidence exists that anoxemia is responsible for the increased pulmonary artery pressure seen following embolization.

The neurogenic role in pulmonary embolism is still unknown, but perhaps vasoconstriction occurs by mechanisms not involving nerve reflexes. J. H. Comroe found by injecting serotonin in the jugular vein of dogs, he could raise the pressure in the pulmonary artery. He postulated chemoreceptors, located in the capillary or venule side of the pulmonary circulation, exist that are sensitive to serotonin. He postulates the breakdown of platelets around the embolus liberates serotonin into the area which activates the chemoreceptors. A. M. Rudolph and M. H. Paul also believe serotonin is a potent vasoconstrictor in the pulmonary bed. It is well known that in anaphylaxis there is intense vasospasm in the lungs of guinea pigs and rabbits. John Irwin observed this to be true with egg albumin in in vivo experiments.

Materials and Methods

Eight to 13 kilogram mongrel dogs, de-wormed, de-loused, and kept isolated two weeks prior to use were anesthetized with sodium pentobarbital, 25 mgm/kgm. Endotracheal intubation was used. A silica carbide, carborundum, grid size 50-90 microns was used. Infusions were made into the right external jugular vein through a 2 millimeter bore polyethylene cannula. The cannula was connected to a "T" tube. To one limb of the "T" tube was attached tubing which drained a 50 cc. reservoir. Physiological saline was used to suspend the 0.5 gm. aliquots of carborundum. Manual stirring was necessary to keep the particles in suspension. The mixture drained into the dog by gravity. Another bottle of saline was connected to the "T" tube and was used to wash the particles out of the tubing into the vein.

All pressure recordings were made on a Sanborn Multi-Channel machine by the use of a Statham strain gauge. Simultaneous electrocardiograms were recorded.

Every seven minutes 0.5 gm. of carborundum was injected into the dogs until they died. Death was defined as the time breathing ceased although the EKG recorded a cardiac contraction for 2-4 minutes afterwards. The time interval of seven minutes was chosen arbitrarily. (From past experience, after this length of time the dogs seemed to stabilize.)

Results

Table III lists the number of the dog, its weight, the number of 0.5 gm. doses infused, the survival time after the first dose, the lethal dose (LD) expressed in gm/kg and the LD as mgm/kg/min. From 1.0 to 5.0 gm. of carborundum was necessary to kill the dogs although the majority died after receiving 2.0 to 3.0 gm. Dog number 227 received 5.0 gm. and lived 63 minutes. He was one of the heaviest dogs weighing 12.9 kg. Dog 233 weighed only 8.7 gm. and was killed with 1.0 gram which was unusual in that weight group.

TABLE III

Dog	*Weight of Dog*	*Number of 0.5 gm. Injections*	*Total Dose*	*Survival in Minutes After 1st Injection*	*LD g/kg*	*LD mg/kg/min.*
227	12.9	10	5.0	63	0.39	6.2
228	11.1	5	2.5	28	0.23	8.2
229	10.2	5	2.5	28	0.25	8.9
231	13.0	6	3.0	35	0.23	6.6
232	10.8	7	3.5	49	0.32	6.5
233	8.7	2	1.0	11	0.12	10.1
234	9.8	5	2.5	34	0.26	7.6
235	10.2	5	2.5	28	0.25	8.9
236	8.6	3	1.5	17	0.17	10.0
237	11.2	4	2.0	27	0.18	6.7
238	11.2	3	1.5	19	0.13	6.9
239	9.8	4	2.0	25	0.20	8.0
240	10.9	4	2.0	27	0.18	6.7

In our series of dogs, the average systolic pressure in the pulmonary artery was 24 mm. of mercury. The range was 10-30 mm. Hg. The average diastolic pressure 10 mm. Hg. The range here was 6-18 mm. Hg. After 0.5 gm. of carborundum there was an increased systolic, diastolic, and pulse pressure. Within zero to five seconds the systolic pressure would rise 10-20 mm. Hg. and the diastolic pressure would rise 5-10 mm. Hg. After 1.0 gm., the systolic pressure would rise 30-40 mm. Hg. over the resting levels with a corre-

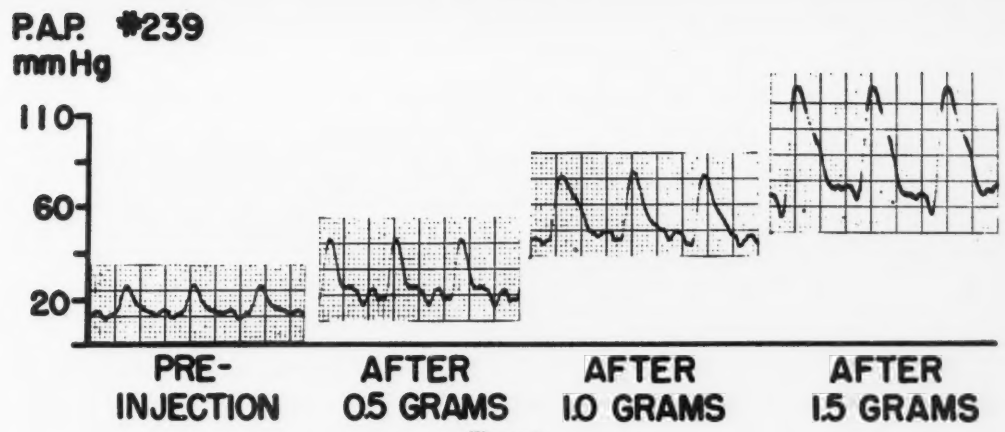


Figure 1

sponding rise of 15-30 mm. Hg. in the diastolic pressure. By the time 1.5 gm. was given, systolic pressures of 90-110 mm. Hg. were consistently obtained along with a diastolic pressure of 50-65 mm. Hg. (Fig. 1). In one animal no increase in pressure was seen after 0.5 gm. of carborundum and 2.0 gm. was necessary to attain a systolic pressure of 90 mm. Hg. Dog 227 tolerated 5.0 gm. of carborundum before dying.

The right ventricle in these dogs could tolerate pressures of 110 mm. Hg. acutely without failure. Addition of more carborundum usually resulted in a fall, rather than a rise, in the pulmonary artery pressure due to failure of the right ventricle. We observed many of the events recorded by others, namely hyperpnea then tachypnea which gradually became so shallow that breathing ceased. This happened without much change in cardiac rate. Cyanosis was present terminally. When the right ventricle began to fail, a gallop rhythm could be auscultated. We failed to observe apnea following injection as Halasz and Marasco did. Heart beats were present two to three minutes after breathing ceased. Electrocardiographic changes included T wave inversion, S-T segment depression and ectopic beats from multiple foci in the right ventricle.

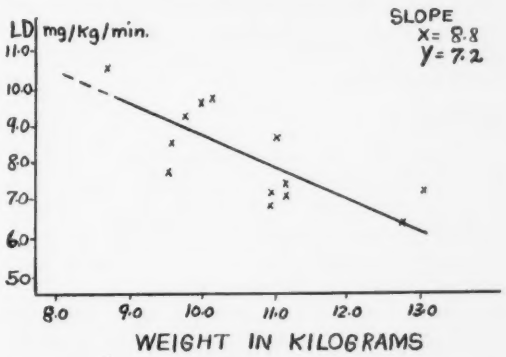
At necropsy, the right ventricle was dilated and the lungs were pale with areas of atelectasis and congestion. Careful inspection of the lungs revealed carborundum diffusely scattered throughout all lobes of both lungs. The lobar and segmental vessels were free of emboli except in two animals where one lobar artery was filled for several centimeters with large clumps of carborundum. These particular instances were probably due to difficulty in achieving even, rapid flow when the aliquots were infused.

If the weight of the dogs in kilograms is plotted against the lethal dose expressed as milligrams per kilogram per minute (see Graph 1), one sees a tendency for the lighter dogs to survive relatively greater doses than the heavier dogs.

The mean lethal dose in mg/kg/min is 7.8. The standard deviation is 1.3.

Discussion

An assumption that we made in the experiment was that the weight of the lung is directly proportional to the total weight of the dog. This is probably not true. While all of the animals were in a good state of nutrition, there was a significant variation in the amount of body fat. It is doubtful if the increased fat seen in some is represented by a proportionally



Graph No. 1

greater lung mass. Therefore, dosage is probably closer correlated to lung weight than total body weight. The age of the animal would be an important variable to consider. Due to the aging process, it seems likely that a young adult animal could tolerate more than an older adult of equal weight. The possibility of species variation should also be considered. Another experiment could be devised using the above factors. The results, interpreted conjointly with the

anatomy of the pulmonary circulation, would be helpful in understanding the altered physiology produced by pulmonary emboli. We could more intelligently study the role of reflex and mechanical mechanisms involved.

Carborundum proved to be a very effective agent in producing acute pulmonary hypertension. We were able, also, to produce chronic pulmonary hypertension in dogs that are still surviving. As was stated in the historical review, the relationship between mechanical or reflex factors is not clear. W. S. Miller estimated that the lungs contain 192,000 arterioles from which all capillaries are formed. These vessels are 165 microns or less in diameter. The size of our emboli were 50-90 microns which were small enough to pass through these vessels but too large to pass through an ordinary capillary. Brenner and Miller, in the study of the anatomy of the blood vessels in the human lung, believe the pulmonary arterioles, unlike those in the systemic circulation, are not well suited for the active control of vascular resistance. The latter contain in the media a thick muscular layer which is lacking in the former. If the particles occlude vessels distal to the arterioles, it seems that in the arterioles the pressure would be increased. We cannot say from this experiment that this increased pressure acts by some means to cause contraction of other patent arterioles. Griffin, Essex, or Knisley have studied this experimentally and deny existance of mechanisms whereby vessels in one portion of the lung are contracted by the presence of emboli in other portions. While reflexes may play a role in, as yet, an indefinite way, the death in these animals could be explained on the basis of a mechanical deterrent to blood flow through the lungs.

Summary

An experiment was performed in which 0.5 gm. amounts of carborundum particles 50-90 microns in diameter were given intravenously at seven minute intervals to 13 dogs until death occurred. Pulmonary artery pressure and electrocardiograms were taken. The mean lethal dose of carborundum was 7.8 mg/kg/min. After a pulmonary pressure of about 90-110 mm. Hg. systolic was reached, the dogs died.

Acknowledgements

This work was done under the direction of Dr. Fred Kittle, Department of Surgery, and Dr. Kurt Reissmann, Department of Medicine, both of the University of Kansas School of Medicine. I wish to thank them for their help as my co-worker, Mr. Luis G. Bianchini, and I performed the experiments.

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Editor's Note: References may be obtained by writing the JOURNAL of the Kansas Medical Society, 315 W. 4th Street, Topeka, Kansas.

Cardiac Resuscitation

(Continued from page 497)

on February 18, 1952.[1] The surgeon was charged with negligence for not attempting cardiac resuscitation and the hospital was charged with negligence for failure to have a stand-by surgeon who could perform cardiac resuscitation. The verdict favored the defendants.

The charge of malpractice comprises two essential allegations:

1. That the physician fails properly to perform his duty.
2. That definite injury to the patient results from this failure.

With the general acceptance by the profession of an established and proven treatment for cardiac arrest and ventricular fibrillation, one must consider the possibility that failure to undertake such resuscitative measures may be more likely to result in a successful malpractice suit against the physician than would an unsuccessful attempt to resuscitate the patient. Similarly, failure of the hospital to have available apparatus now accepted as effective and necessary for treatment of this condition may leave the hospital open to legal action.

While more and more Kansas surgeons are performing thoracotomy in cases of cardiac arrest, most Kansas Hospitals have not accepted defibrillators and other apparatus as essential equipment for the operating room or hospital in general.

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Epigastric Pain, Chills, Fever, Nausea, Weakness, and Loss of Weight

Case Presentation

A 64-year-old white man was admitted to KUMC on February 8, 1958, with a complaint of upper abdominal pain, fever, and fatigue. He died on March 12.

He had apparently been well until one year before admission when he developed nausea, weakness, and fatigue. Three months before admission he noted a continuous, dull, epigastric pain which radiated to the right infrascapular area and which was aggravated by eating. Two weeks before admission he had fever, chills, and extreme fatigue, and it became necessary for him to stop working. At that time he also had an episode of melena and one week later he had a spell of vomiting.

His mother died of cancer of the liver; his father died of "hardening of the arteries."

He had worked as a DDT truck sprayer from 1955 to 1957. He had had dyspnea on exertion and ankle edema for about five years and a chronic, productive cough for about three years. He had had no orthopnea, jaundice, or any change in bowel habits. He had symptoms of prostatism.

The patient was a well developed, obese, white man who appeared to be chronically ill. His blood pressure was 120/85; pulse 76 and regular; and his respiratory rate was 16 per minute. He was edentulous. He had no lymphadenopathy. His chest was clear to percussion and auscultation. The PMI was in the left fifth intercostal space in the anterior axillary line, and there was a grade II systolic murmur over the aortic area which was also heard in the third intercostal space at the left sternal border. The second aortic sound was greater than the second pulmonic sound. There was some guarding in the abdomen. The liver was palpated one and one-half cm. below the costal margin and was questionably enlarged. A tender, non-pulsatile mass measuring 3x4 cm. was palpated over the epigastrium and the right upper quadrant. The spleen and kidneys were not palpable. The prostate was soft and enlarged one plus, but there were no masses. The extremities were normal.

LABORATORY REPORTS

The specific gravity of the urine was 1.014 with two plus albumin and few waxy casts per high power field. Bence-Jones protein was negative. The white count was 14,300 with 84 per cent polymorphonuclears, 81 filamented (3 non-filamented), 15 per cent lymphocytes, and one per cent monocytes. The hemoglobin was 10.8 gm., and the hematocrit was 47 ml. Repeat blood counts showed a persistent leukocytosis with the same differential count. The sedimentation rate was 25 mm. per hour, and the C-reactive protein was three plus.

The VDRL was non-reactive. The blood urea nitrogen was 22 mg. per cent on admission, and a repeat test on March 8 was 43 mg. per cent. The fasting blood sugar was 109 mg. per cent; carbon dioxide, 21.8 mEq/L; sodium, 138 mEq; potassium, 5.4 mEq; and chloride, 94 mEq. On March 6 the sodium was 123 mEq; potassium, 5.4 mEq; and the chloride, 95 mEq. On March 10 the sodium was 130 mEq; potassium, 7.1 mEq; chloride, 101 mEq; carbon dioxide, 20.3 mEq; calcium, 5.3 mEq; phosphorus, 2.1 mEq; albumin, 3.7 gm. per cent; and globulin, 3.3 gm. per cent. Paper electrophoresis showed albumin 35 per cent; alpha-1 globulin, 11 per cent; alpha-2 globulin, 19 per cent; beta globulin, 14 per cent; and the gamma globulin, 22 per cent. The total cholesterol was 133 mg. per cent (60 per cent esters). The alkaline phosphatase was 0.4 units; thymol turbidity, 3 units; total serum bilirubin, 0.7 mg. per cent (direct, 0.4 mg. per cent); amylase, 45 units; lipase, 0.4 units; serum iron, 22 mcg. per cent; bromsulphthalein excretion, 37 per cent; transaminase, 14; and prothrombin time, 83 per cent. The blood ammonia

Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center, as well as by the third and fourth year classes of students.

Figure 1. Electrocardiogram taken on March 6, 1958.

was 150 mcg. per cent. A repeat alkaline phosphatase on March 5 was 3.9 units.

The venous pressure was 16 cm. The arm to lung circulation time was 11 seconds and the arm to tongue time was 15 seconds.

A gastric analysis showed an absence of free acid in the fasting specimen, but after stimulation with betazole hydrochloride the highest value was 50 degrees. Fecal tests for occult blood were negative. The phenolsulphthalein excretion was 8.5 per cent in the first fifteen minutes and 31.4 per cent in one hour. A skin test for histoplasmosis was positive and for tuberculosis, negative.

On March 12 a sputum culture showed coagulase negative hemolytic and non-hemolytic Micrococcus aureus. The urine culture was negative.

On admission the patient had a temperature of 102 degrees. Two days later he was afebrile, but fever developed again on his sixth hospital day. He went to surgery on February 19, after which his postoperative condition was satisfactory. One week later a morbiliform rash was noted over his trunk and extremities. This was more prominent in the axillary and crural areas but was not associated with any itching. On February 4 he complained of shortness of breath, and the next day there was a sudden drop in his blood pressure. On March 12 at 6:15 p.m. he vomited a small amount of blood, and he continued to vomit larger amounts at frequent intervals. He went into shock, and died one hour later.

Dr. Mahlon Delp (moderator): Are there any questions?

Orville Boicourt (student)*: Did he have a history of colicky pain in the right upper quadrant?

Dr. Delp: He had abdominal pain when his body was jarred, and he had pain and discomfort after eating. The chest pain radiated through to the infrascapular area when he stooped over.

Albert Duncan (student): Were lymph nodes palpable in the axillae?

Dr. Delp: No, but there was some question of an abdominal mass because of the patient's guarding.

Ashford Galbreath (student): Was Murphy's sign present?

Dr. Delp: I do not believe that pain was elicited.

Kenneth Kihle (student): What was his intake and output?

Dr. Jose Martins (resident in medicine): After surgery the highest intake was 2800 ml., and the output was about 1500 to 2000 ml.

Orlyn Engelstad (student): Had he had an appendectomy?

Dr. Martins: There is no record of it.

Don Van Derby (student): Was a glucose tolerance test done?

Dr. Martins: No.

Mr. Kihle: Was he given a diuretic before admission?

Dr. Delp: No, but about four or five years ago he apparently had had some edema which did not disappear spontaneously.

Mr. Boicourt: When was the blood ammonia level recorded?

Dr. Delp: Three days before death.

Wilbur Neel (student): Had he been employed in any way other than as a DDT truck sprayer?

Dr. Martins: He had worked for several years as a mechanic for the highway department, and previously he had worked as a smelterer in southeast Kansas.

Mr. Galbreath: What were his smoking habits?

Dr. Martins: He had not smoked since January, 1958, but before that he smoked thirty cigarettes a day.

Don Ginardi (student): Was there a bilirubin subsequent to the one recorded in the protocol?

Dr Martins: Just before death the total bilirubin was 1.5 mg. per cent, direct 0.1 mg. per cent.

Donald Hayen (student): Had he had any previous transfusions?

Dr. Martins: No, he had not.

Mr. Duncan: What was his temperature course?

Dr. Martins: He had had chills and fever for several days before admission. He had a slight fever before surgery, but afterward his temperature was normal. By the end of February he again had a slight fever, but he became afebrile during the first week of March.

Dr. Delp: If there are no more questions, we will have the electrocardiograms.

Mr. Hayen: An electrocardiogram taken on Feb-

* Although a medical student at the time of this conference in November, 1958, he, like the others referred to as students, received his M.D. degree in June, 1959.

ruary 10, 1958, shows P waves and a normal sinus rhythm of 80. The QRS duration is at the upper limits of normal, but otherwise I interpret this as a normal tracing.

On March 6 the tracing (Figure 1) again shows a sinus rhythm and a rate of 160. There is a slight terminal depression in the S-T segment in leads I, aVL and V_6. I interpret this tracing as sinus tachycardia and possible left ventricular ischemia.

Dr. Delp: Do you believe that this tracing will contribute anything to the diagnosis?

Mr. Hayen: No, I do not.

Dr. Delp: We will now have the x-rays.

Mr. Ginardi: No bony abnormalities are seen in the P-A film of the chest (Figure 2). The lungs are well expanded, and there is no atelectasis. The dome of the diaphragm is elevated on the right side, but this is probably normal. There is some flattening of the diaphragm on the left which could indicate emphysema. I believe the overall picture of the x-ray is normal except for some emphysema and possible elevation of the right diaphragm and flattening of the left.

In the lateral film of the chest there is lipping of the spine indicating osteoarthritis. I believe the heart is normal. Again, there is a superimposed elevated diaphragm on the right and flattening on the left. In the posterior lung fields there is increased marking compatible with emphysema.

The x-ray taken at the time of operation shows the gall bladder filling normally and not enlarged. The dye can be seen going into the liver and the duodenum with no blockage.

The film of the barium enema shows a deflection of the hepatic flexure which I interpret as evidence of an enlarged liver. It is apparent that the patient had had intravenous pyelograms. We can see filling of the ureters, and there is a normal pattern of the calyx on both sides. No abnormalities of the kidneys are noted.

The next film shows anterior displacement of the stomach. There is good filling, and I believe there is a normal rugal pattern. Subsequent films show some hepatic enlargement and displacement of the stomach.

Dr. Delp: Dr. Youngstrom, may we have your comments, please?

Dr. Karl Youngstrom (radiologist): The dome of the diaphragm on the right is that of a pattern usually referred to as a "bosselation," and it is frequently seen in older people with weak diaphragms. It is, here, consistent with enlargement of the liver, especially when it bulges anteriorly. I believe, however, that it is more consistent with bosselation of the diaphragm; in other words, a weak place allows the liver to bulge slightly whether it is enlarged or not. Hepatic enlargement is indicated by the extension of the liver down to the crest of the ileum. The student must be complimented for recognizing that the stomach is displaced anteriorly. The flattening of the diaphragm on the left is consistent with some emphysema which is fairly common in people of that age with degenerative chest changes.

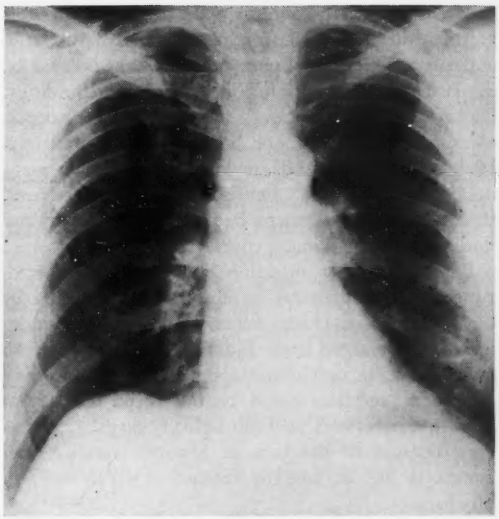


Figure 2. X-ray of chest on February 10, 1958.

Dr. Delp: Of particular interest is the history of chills and fever, the equivocal mass in the abdomen, the unusual pain and the hematemesis. Apparently he had had some bleeding before he was admitted, as it was reported that he had a tarry stool.

May we have the differential diagnosis now, please?

Differential Diagnosis

Mr. Duncan: Our case today is that of a 64-year-old white laborer who came here complaining of upper abdominal pain and progressive nausea, weakness, and fatigue of about one year's duration. Three months before admission he developed epigastric pain which was accompanied by chills, fever, vomiting, and rapid weight loss. On admission a tender, small mass was noted in the epigastrium, and the liver was slightly palpable. Laboratory tests showed a persistent leukocytosis with a high polymorphonuclear count and a hypochromic anemia. Bromsulphthalein and phenolsulphthalein tests were abnormal. There were albumin and casts in the urine. There was a low serum iron and a high blood urea nitrogen level. The sedimentation rate was fast. Eleven days after admission a surgical procedure was performed. His postoperative condition was satisfactory for about two weeks when he began to develop shortness of breath, and there was a sudden drop in his blood pressure. One week later he had an episode of hematemesis which resulted in shock and death.

My differential diagnosis will be based on epigastric pain and rapid weight loss. I shall first rule out pancreatic cyst and perinephric abscess because of the lack of appropriate history and physical findings. Because of his previous history of melena, peptic ulcer perhaps should not be ruled out completely. Multiple myeloma and amyloidosis should be mentioned because of the multiple system involvement, but I shall dismiss them because of the laboratory findings and the clinical course.

Diseases due to infection should be considered. Amebic and pyogenic liver abscess are suggested by the leukocytosis, chills, fever, the palpable, tender mass, and enlarged liver. Elderly people can have an acute, subacute or chronic appendicitis with varying symptoms, and this could be the focus for a pyogenic liver abcess. That is an unlikely diagnosis, however, because of the lack of history, jaundice, distention or ascites, and the absence of sepsis early in his disease.

Acute and chronic cholecystitis are extremely dangerous gangrenous forms which vary from mild to catarrhal types. The presenting symptoms may be severe pain, muscle spasm, rebound tenderness, nausea, and vomiting. Stones are present in about 90 per cent of these cases, and there may be jaundice and leukocytosis. I shall rule this out, however, because of the patient's protracted course, the absence of stones and any pre-existing cholecystitis.

Subphrenic abscess is an unlikely diagnosis because of the lack of depressed respirations and the basilar pneumonia so frequently found in that disease. Furthermore, the x-ray of the diaphragm is not compatible with it.

Subacute and acute bacterial endocarditis must be mentioned because of the patient's fever, murmur, and anemia, but I shall dismiss it as a possible diagnosis because he had no constant fever or other signs such as change in the heart murmur, Osler's nodes, splinter hemorrhage, splenomegaly, or clubbing of the fingers.

Chronic relapsing or calcerous pancreatitis can be ruled out because he had no acute attack, no severe colicky pain, or greasy, foul-smelling stools. In 50 per cent of these cases calcification is demonstrated by x-ray.

MALIGNANT DISEASES

Carcinoma of the lung must be considered because of his dyspnea on exertion for five years, the chronic cough for three years' duration, and his history of smoking one and one-half packages of cigarettes a day. I shall rule that out, however, because of the lack of hemoptysis and the duration of symptoms. Some carcinoma of the lungs can be metastatic, however, with no primary located at that time.

Cirrhosis is a predisposing factor in about 50 per cent of patients with primary carcinoma of the liver. Other factors which make that an unlikely diagnosis are the lack of jaundice, the normal alkaline phosphatase, and the absence of ascites.

Carcinoma of the stomach is suggested here because of the insidious onset of symptoms, the weight loss, vomiting, anemia, and the low serum iron. It is an unlikely diagnosis, however, because of the lack of blood in the vomitus, and there were no x-ray changes in the stomach other than forward displacement.

Malignant lymphoma and retroperitoneal lymphomas must be considered because of the history of fever, weight loss, nausea, and epigastric pain. In retroperitoneal lymphoma the stomach is displaced anteriorly. Secondary involvement of the genitourinary tract with albuminuria occurs in about 20 per cent of these cases. However, there is often a generalized lymphadenopathy and splenomegaly at some time during its course, and I shall, therefore, rule out that diagnosis.

Carcinoma of the gall bladder is found in 4.5 per cent of all carcinomas, and occurs most often in persons of middle and advanced age, about 70 per cent occurring in women and 30 per cent in men. Gallstones precede carcinoma in about 50 per cent of these cases. Pain in the right upper quadrant and in the right subscapular area is usually the most frequent symptom and occurs in 90 per cent of the cases. This pain may be typically gall bladder colic, but it is usually more severe. A weight loss of 10 to 15 pounds occurs in 65 per cent of these cases. A tender mass is often found in the right upper quadrant. If liver metastases have occurred they are often palpable as tender nodules in the lower liver edge. There is jaundice in only about 25 per cent of the cases, and, as a rule, it is progressive and persistent. Anorexia, nausea, and vomiting are common symptoms as the disease progresses. Metastases occur early and extend first to the liver and then to the lymph nodes and surrounding organs. Various degrees of infection are invariably present, and range from localized infections surrounding the tumor to acute cholecystitis or ascending cholangitis due to blockage of the cystic or common duct by stones or tumor. This is an unlikely diagnosis, however, because there was no previous history of stones, and the gall bladder functioned normally.

My final diagnosis is carcinoma of the pancreas which accounts for about two per cent of all carcinomas. It usually occurs in the 40 to 50 year age group, and about three times as frequent in men as in women. The course of pancreatic malignancy is usually rapid, and most cases terminate in less than one year. The symptoms most frequently reported are pain, weight loss, jaundice, nausea, vomiting, weakness, fatigue, constipation, personality changes and multiple venous thromboses. The most striking symp-

tom is the weight loss which may be as much as 80 pounds. The second most significant symptom is the pain which is extremely variable and can be misleading in that it may mimic biliary or intestinal problems. The pain is located in the upper abdomen and radiates through to the back. It is dull and often severe and is usually more intense following meals with no relief from physical activity or from bowel movements. When the patient lies on his back the pain becomes worse, and the jackknife position is usually assumed.

Approximately 97 per cent of patients with carcinoma of the head of the pancreas have jaundice, but it is infrequently seen in carcinoma of the body and tail. Laboratory findings are not diagnostic for jaundice, but the elevated bilirubin, the glucose tolerance curves, the determination of pancreatic enzymes from the duodenum, and stool examinations for excess fat or protein all contribute in making the diagnosis of carcinoma of the pancreas. I believe that our patient had carcinoma of the body and tail of the pancreas because of his history of epigastric pain which radiated to the back, the rapid weight loss, nausea, vomiting, extreme fatigue, and the absence of jaundice. The physical findings of a tender upper abdominal mass and the elevated fasting blood sugars support that diagnosis. The problem of an apparent septic course could tend to refute it, but in neoplastic diseases fever, chills, sweats, and leukocytosis with shift to the left are frequently noted. Carcinoma of the pancreas is one of the neoplasms known to cause this. The clinical course is only about one year's duration. Approximately three months before admission the patient had an acute exacerbation, and his condition rapidly became worse. Two weeks before admission final sepsis appeared, but he later became afebrile. His temperature rose again on the sixth, seventh, and eighth hospital day, and he had fever terminally. The productive cough, dyspnea, ankle edema, cardiac enlargement, increased venous pressure, prolonged arm to lung time, normal lung to tongue time, albuminuria, casts and decreased bromsulphthalein, and phenolsulphthlein excretions can be explained on the basis of pulmonary emphysema. He also had some myocardial ischemia. The low hemoglobin with reduced serum iron concentration is consistent with iron deficiency anemia, and this, together with the low serum iron, is certainly consistent with a neoplasm and infection. I do not believe that the bleeding he had before he entered the hospital could account for his anemia.

A surgical procedure was performed on the twelfth hospital day following which he did well for thirteen days and then developed shortness of breath and hypertension. There was transitory improvement, but he began to vomit blood, went into shock, and died. I believe the shock and bleeding were due to carcinoma of the pancreas eroding into the posterior aspect of the stomach or the duodenum. A retroperitoneal lymphoma with erosion and hemorrhage could also involve the stomach posteriorly. Gram negative bacteremia can also cause shock and gastrointestinal bleeding and is frequently seen in older, debilitated patients. This may persist for some time and is associated with considerable weight loss, fever, chills, and severe shock. Death usually occurs from twelve hours to twelve days after the initial onset of shock. Pulmonary emboli could have caused the patient's dyspnea and hypertensive episodes, but he had no hemoptysis or pleuritic chest pain.

Clinical Discussion

Dr. Delp: Thank you. What is your diagnosis, Mr. Hayen?

Mr. Hayen: Carcinoma of the body and tail of the pancreas.

Dr. Delp: Why was the patient bleeding?

Mr. Hayen: He was probably bleeding terminally because of involvement of the posterior aspect of the stomach.

Dr. Delp: Mr. Hardy?

Mr. Hardy: The bleeding was probably due to erosion by the carcinoma.

Dr. Delp: Mr. Boicourt?

Mr. Boicourt: He could have had acute thrombosis of the splenic vein with development of esophageal varices.

Dr. Delp: How do you account for his fever, Mr. Galbreath?

Mr. Galbreath: Fever, chills, and a high white count are common findings in carcinoma of the pancreas.

Dr. Delp: Mr. Engelstad, how did you rule out inflammatory disease of the gall bladder?

Mr. Engelstad: This was ruled out because of his two-week history here, and because there was no history of stones.

Dr. Delp: Mr. Hayen, can you explain the leukocytosis?

Mr. Hayen: Leukocytosis is present in carcinoma of the pancreas.

Dr. Delp: Mr. Ginardi?

Mr. Ginardi: I believe there was an infection.

Dr. Delp: Did his tenderness in the right upper quadrant affect your diagnosis, Mr. Duncan?

Mr. Duncan: No, it did not.

Dr. Delp: Dr. Klotz, what was your diagnosis?

Dr. Arthur P. Klotz (internist): My diagnosis before surgery was a cholecystic abscess.

Dr. Delp: Dr. Friesen?

Dr. Stanley Friesen (surgeon): My preoperative diagnosis was chronic cholecystitis and pericholecystic abscess. The chills, fever, and tenderness in the right

Figure 3. Gross photograph of the tumor in the tail of the pancreas. The tumor mass has been bisected. The capsule of the spleen can be seen in the background.

upper quadrant gave the impression of an inflammatory lesion.

Dr. Delp: Dr. Klotz, what was your impression of the patient's bleeding?

Dr. Klotz: My impression was that it was probably agonal and represented bleeding from an erosive esophagitis.

Dr. Delp: Thank you. We will now have the pathologist's report.

Pathological Report

Dr. Peter Rasmussen (pathologist): At autopsy there were evidences of cardiac failure with bilateral hydrothorax, ascites, ankle and pretibial edema, and acute pulmonary edema. A considerable amount of blood and mucus was present in the bronchi.

A rounded primary tumor replaced the tail of the pancreas (Figure 3), and the mass, yellowish-white, and somewhat lobulated in appearance, measured 3 cm. in diameter. In addition, porta-hepatic lymph nodes were infiltrated by tumor tissue.

Microscopically the cancer in the pancreas varied in morphology (Figure 4). Some portions contained large masses of tumor cells often simulating macrophages, but sometimes forming abortive acini. Mitoses were plentiful. A granular amorphous pink material could be seen in some of the larger tumor spaces.

A reticulum stain confirmed that tumor nests tend to be clumped or surrounded by areas of reticulum rather than individual tumor cells surrounded by reticulum, thus indicating a carcinoma. In addition, periodic acid-Schiff stains demonstrated mucus production by occasional neoplastic cells. The appearance, then, was that of an anaplastic carcinoma. Microscopic metastases could be found in the lungs.

The body of the pancreas was replaced by irregular acini. The pattern was not like that seen before, but was one of chronic pancreatitis with extensive, acute and chronic inflammatory infiltration as well as interstitial fibrosis.

There was interstitial edema in the kidney with some dropping out of nuclei and tubular degeneration in proximal convoluted tubules. In addition, focal thrombosis of arcuate veins and occasional foci of acute and chronic inflammation were evident. These findings were most compatible with an acute toxic nephrosis following infection or debilitation. The elevated blood urea nitrogen of 44 mg. per cent may not have been associated with this more terminal nephrosis, but probably was associated with a concomitant low serum sodium and represented a prerenal type of azotemia.

An acute pericholangiohepatitis with infiltrates of

(Continued on page 531)

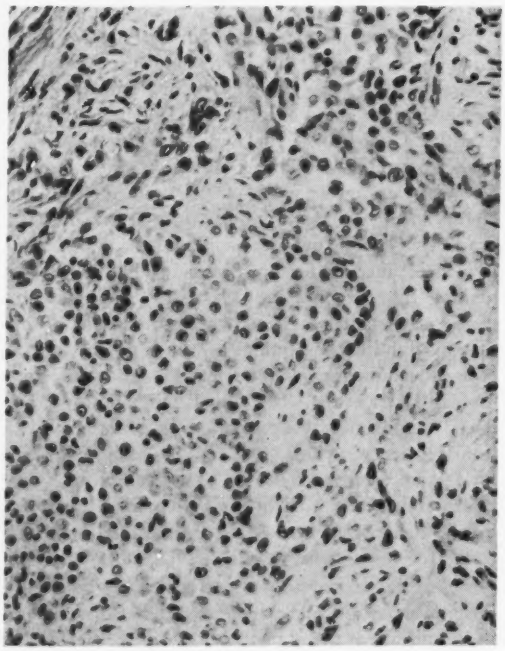


Figure 4. Photomicrograph of the main tumor mass. No organoid pattern can be seen. Hematoxylin eosin stain. 150X.

The President's Message

DEAR DOCTOR:

Your indulgence is sought for using this page again as a forum for a discussion of the Forand Bill.

This bill, H. R. 4700, is only one of many of a similar nature introduced into the past session of Congress and that will undoubtedly be brought up for committee or floor action at the second session of the 86th Congress. We who believe that this type of legislation is undesirable should be able to discuss the merits and demerits of this bill with our patients, friends, and political representatives.

As you know, the Forand Bill is an amendment to the social security act providing for hospital, surgical, and nursing home care to beneficiaries of social security, mostly those over age 65, 16 million U. S. citizens. This is to be financed by an increase in social security taxes, which are already scheduled to reach 9 per cent of payroll, up to $4,800.00, in the next few years.

Organized medicine is opposed to this legislation because:

1. The care of the aged is a community problem, best solved by the cooperative effort of doctors, hospitals, nurses, insurance companies, and responsible community leaders, not a federal government controlled plan.
2. This is a political approach to a health problem developed by non-medical people.
3. Government intervention will weaken the patient-physician relationship.
4. Political abuses and administrative wastes would be predictable.
5. Costs would be excessive.
6. Over utilization of hospitals can be expected.
7. The patient would not have unlimited choice of physician or hospital.

Medicine's answer to the problem of the care of the aged are beginning to crystalize. The AMA, the Blue plans, and the Health Insurance Council are exploring means of providing voluntary health insurance at a price the aged can afford. Many such plans are in effect and are meeting a need successfully. Conferences are being held to explore the entire needs of the aged, not just the health needs. The AMA has backed legislation aimed at assisting in the construction of nursing homes and convalescent centers. It is interested in experiments with progressive hospital care, home nursing, and home maker services.

Much has been accomplished to meet this new challenge to medicine. You can do your part by being familiar with the problem and making your thoughts known.

Fraternally,

Glenn R Peters M.D

President

Just BROWSING

The use of mercurials, so long considered indispensable in therapeutics, was certainly not in the best favor of Dr. William J. Burge of Atchison, when he gave the "Report of the Committee on Mercurial Preparations" at the 1872 meeting of the KMS. A few excerpts from this report will demonstrate that he not only had strong feelings, but seemed to have a basis for them.

". . . It must be apparent to the most cursory observer, that within the last twenty-five or thirty years the practice of medicine has undergone very marked modifications. The lancet, for example, which within the memory of many of us, was in the hand of every physician, and in the arm of every patient, has finished its course and been laid away in the grave as quietly as many of its former victims. . . . Gradually it was discovered that more patients recovered who were not bled than of those who were, and this was true in every variety of disease. It took practitioners a long time to learn this, but they *have* learned it, so thoroughly that today we hear very little said about bleeding. With regard to mercurials, . . . men who were in the habit of prescribing calomel with a high-hand and with a stretched-out arm for almost every patient, now handle this potent remedy with gloved fingers, or only pick it up at all, as a dernier resort. The general and indiscriminate use of the drug has been fraught with so much disappointment that many physicians as well as patients have concluded that the little god in whom they trusted, is quite as likely to salivate as to save.

"We do not deny that mercurials are powerful remedial agents . . . but that they are indispensable to the successful treatment of any disease, admits of the greatest doubt. In fact, . . . thousands of intelligent physicians in every part of the world are daily demonstrating that any disease which can be cured by mercurials, can as speedily be cured by other and safer remedies. . . .

"At the period when mercurials were introduced to the profession, chemistry was in its infancy, and the materia medica from which the formulas of the wisest physicians were drawn consisted of little else than a mass of crudities, incongruities, and absurdities. For the comparatively few remedies which are still counted worthy of a place in our pharmacopoea we feel duly grateful to those good old hard-working experimentors, observers, and accumulators, but we nevertheless rejoice in the light of the past few years which has discovered so much rubbish among the valuables, and so many valuables among the rubbish. May we not hope that with a little more sweeping and garnishing our armamentarium medicarum will be fit for the closest inspection. Let us beware, however, lest in our zeal to get rid of the chaff, we lose some of the wheat also. Rich as we undoubtedly are in remedies, we cannot afford to part with any of our treasures. If it can be proved that any preparation of mercury is more positively indicated in any given diseased condition, than other remedies with which we are acquainted, far be it from me to discourage its use. . . . Whatever benefits may have accrued to the human race from the introduction of mercurials into medical practice, it has certainly suffered a vast amount of evil on account of the ignorant and indiscriminate use of these remedies. For every intelligent physician who prescribes a dose of calomel, there are a thousand self-constituted doctors who deal it out as freely as they would chalk or cheese. . . . The people pick their panaceas from the remedies which they observe to be most frequently used by the doctors. This accounts for the wide-spread employment of mercurials among non-professional persons among the middle and poorer classes. The evils resulting from the abuse of these remedies have continued beyond the period of necessity for their use. In skillful hands they were undoubtedly at one time the best known weapons wherewith to combat disease. We believe that time has passed. . . . I believe the profession today knows very little more about mercurials than it did twenty years ago, except the simple fact that they are not indispensable to the cure of any human malady."

How long do you suppose it will be before similar statements are made about antibiotics, adrenocortico steroids, the surgical removal of cancer, or its treatment by radiation?—O. R. C.

Editorial COMMENT

Cost of Health Care

Illness is an unexpected blow, not too unlike the panic a person feels when he turns a switch and the lights fail. The shock of the unanticipated disrupts his plans, alters his schedule and spreads resentment.

The cost of health care is so directly related to the emotional effect of illness, to the patient's sense of loss, his inadequacy, in addition to the physical side of the problem that these bills often become tangible factors against which a significant opposition may be raised.

The target is of course the physician. So logical is this warped sequence of reactions that it might be added to an impressive list as a side-effect of illness. It could almost be classified as an unwelcome by-product the doctor accepts with his professional career. It represents a penalty of leadership which only excellent service and the most ingenious tact has controlled to its present degree.

Not that the public lacks confidence in medicine. The layman speaks glibly about miracle drugs and about advancements in surgery. He has accepted these with a faith out of all proportion to his understanding. He looks upon health care as he does his mathematics in which the answer is predictable. Variations, of which the physician is so painfully aware, are problems the layman does not choose to recognize.

The fact this subject is complex makes it more difficult but if the physician is unable to explain his status as it relates to the patient and his illness, to the patient and his hospital, his drugs, his professional care, as well as to the patient and his emotional stress, how may he receive this understanding? Who other than the physician can show him what he receives for each dollar he invests in health? It appears a stronger voice than has yet spoken is needed at this time. Perhaps one will be found with the inspiration for words that can touch the layman's understanding.

How this is to be stated depends upon his genius but the subject can be anticipated. For example, the added life span may well be the most dramatic single historical fact of the past fifty years. Its social and economic implications exceed the bounds of imagination but the impact of longer life is of even vaster personal significance, Figures state life expectancy during the Roman Empire to have been 29 years, to have risen in 1900 in America to 49 years, and today to have passed 70 years, and is still climbing. Those are the cold facts. They need the warmth, the humanity of translation into individual meaning.

It is not enough to catalog medical advancements as one announces the new model of an automobile. The latter is an achievement of its own, the end result, but everything new in health care represents only a tool that lies dormant and without purpose until human skill adapts its service to a human life in need. There is a difference, which needs expression.

The economics of health care also cry for better understanding. Most laymen realize hospital costs have risen but few carry an appreciation of this fact. It is not enough today that the public learns physicians receive less from the present health care dollar expenditure than in 1929. In that year 32.6 cents of each dollar spent in health services went to the doctor. In 1957 this was 24.5 cents. Dentists' services take less also, 16.4 cents in 1929 and 11.3 cents in 1957. The drug percentages remain constant during this period at 20.6 cents for each of the two years studied. Several minor items fluctuate but their status does little to influence the picture. It is hospital cost, rising from 1929 when 13.7 cents of each dollar was spent at hospitals to 1957 when the take was 25.8 cents, that is important.

Those are the statistics but they stand poles apart from the individual who finds a debt on his hands. His becomes a personal problem and only the well integrated or the well informed can view this objectively. So, after all, it becomes the responsibility of the physician to interpret this information into areas of the patient's personal experience. Where this has been successfully accomplished medicine is not criticized. The need remains, however, for an increased effort to tell this story honestly and well, to tell it with dignity and understanding of the listener's needs. This can be done. It remains now to find such

voice. Who could do it better than the doctor as he visits with his patient during the course of his professional service?

Letter From AMA

The Forand Bill, listed as H. R. 4700, will, in 1960 as it was in 1959, be the principal weapon employed in the battle to socialize the practice of medicine in the United States. Students of political science predict a major effort will be conducted over this issue during the second term of the 86th Congress convening on January 7, 1960. The bill currently rests in the Committee on Ways and Means in the House of Representatives.

In brief this amendment to the Social Security Act would pay from that fund the cost of surgery, hospital care up to sixty days and care in a nursing home up to 120 days for persons entitled to retirement benefits under Social Security and their dependents. It would increase the social security tax.

Hearings were held on this bill last July and many arguments were presented for and against the proposal. Those asking for the passage of the Forand Bill declare the aged cannot afford the cost of health care and are being neglected. Medicine and many in other areas of activity deny this as an unfounded statement, declare the measure socialistic and economically unsound.

The physicians of Kansas exerted some considerable effort both in assuring that the aged in this state were cared for and in acquainting their Congressmen of the dangers in the Forand Bill. Even more activity will become evident in this field during the next few months.

A letter from F. J. L. Blasingame, M.D., executive vice president of the American Medical Association expresses thanks to the doctors of Kansas for their efforts. The Council directed this should be made known to each physician. It is therefore published here.

American Medical Association
535 North Dearborn Street
Chicago 10, Illinois
July 30, 1959

If time would permit, I would like very much to write individual members of the Kansas Medical Society as well as members of lay organizations in your state, to thank them for the splendid cooperation they gave us during the past month in regard to the Forand Bill. Your personal effort in cooperating with Mr. Dalbec of our Field Service Division in alerting the profession was most gratifying.

The spontaneous results of the many letters, telegrams, etc., received by your Congressmen; the excellent written testimony from your Society and the presence of Glenn R. Peters, M.D., in Washington; the most timely letter from Mr. Carl Lamley and Sister Mary Roberta, R.S.M.; were most encouraging to the staff of the American Medical Association. All this, as well as the cooperation indicated by lay organizations in your state, represents the only possible way we have of winning this fight.

Would you kindly convey our thanks to all concerned and may we earnestly solicit your continued cooperation in keeping your Congressmen informed on all legislation pertaining to health when they are at home or in Washington.

This indeed will preserve the cause of freedom, and, for this philosophy, we cannot be challenged.

Sincerely yours,

F. J. L. Blasingame, M.D.
Executive Vice President

Healing Arts Practice

The Council of the Kansas Medical Society in a meeting at Topeka on October 4, 1959 received the following correspondence. It was considered of sufficient significance to the physicians of this state that action was taken directing it to be published in the JOURNAL.

TO WHOM IT MAY CONCERN:

At the July 30, 1959 meeting of the KANSAS STATE BOARD OF HEALING ARTS, the following Resolution was adopted:

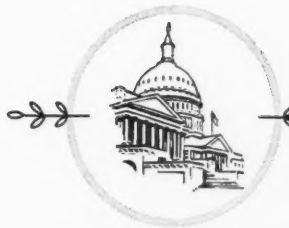
RESOLUTION

WHEREAS, the Kansas State Board of Healing Arts has been advised that some of the hospitals in the State of Kansas may be engaging in the unauthorized practice of the Healing Arts.

WHEREAS, the Kansas State Board of Healing Arts is charged by law with the regulation and control of the practice of the Healing Arts in the State of Kansas.

NOW THEREFORE, be it resolved that the Kansas Hospital Association, the Kansas Medical Society, the Kansas State Osteopathic Society, the Kansas Chiropractors Association and Kansas Chiropractic Society, the Kansas Physicians Service, the Kansas City Blue Shield, and the insurance companies writing health and accident and compensation insurance, be notified that hospitals in Kansas are not authorized to engage in the practice of the Healing Arts as defined by the Kansas Healing Arts Act, and that all professional services performed in the practice of the Healing Arts must be performed by or under the supervision of a duly licensed practitioner.

F. J. Nash, M.D., Secretary
KANSAS STATE BOARD OF HEALING ARTS
364 New Brotherhood Bldg.
Kansas City, Kansas



Washington HIGHLIGHTS

This summary of Washington news is prepared by the A.M.A. Washington Office for distribution to state medical journals.

Chamber Warns of Forand Fight

The U. S. Chamber of Commerce and two key Congressmen, all opponents of the so-called Forand bill, recently issued separate warnings that an all-out effort will be made to get the controversial legislation through Congress next year.

In its weekly report to members, the Chamber predicted there will be "a powerful attempt" in the next session of Congress to enact the bill (H. R. 4700) which would increase social security taxes to help pay for the cost of the Federal government providing surgical and hospital care for social security beneficiaries.

The Chamber warned that passage of the legislation would mark a major break-through into the welfare state. It probably would lead to a compulsory Federal program providing complete medical care for everyone, the Chamber said.

There would be "no stopping" of such a program once it got started, the report said.

The Chamber called upon communities to find orderly solutions to the problems of the aging. Otherwise, solutions will surely be imposed from Washington, the report added.

Congressmen Urge Doctors to Act

Similar warnings were voiced by Reps. Richard M. Simpson (R., Pa.) and Thomas B. Curtis (R., Mo.), key members of the House Ways and Means Committee where the bill was put on the shelf last session.

Rep. Curtis urged that the medical profession and other leading opponents make a strong counter-drive in an all-out effort to block passage of the bill next session. Unless there is such action, he said he would have to "regretfully" predict that legislation along the lines of the pending bill probably will be enacted in 1960.

Rep. Simpson said that H. R. 4700, and similar legislation affecting the medical profession, "make it imperative that every doctor keep informed on legislative issues before Congress." He also urged that physicians "become patriotic political forces" by giving "their informed viewpoint" to lawmakers at all levels of government.

Rep. Simpson said it "is important" that opponents of H. R. 4700 develop "appropriate alternatives" to solve the health care needs of the aged.

He promised to continue to cooperate with the medical profession to guard against the disastrous consequences of compulsory national health insurance.

High Hopes for Keogh-Simpson Bill

House Democratic Leader John McCormack of Massachusetts expressed hope that Congress next year will stamp final approval on another bill of particular interest to physicians. He praised the Keogh-Simpson bill (H. R. 10) as "meritorious legislation" and said it "should be enacted into law next year." The measure, which was passed by the House last spring but left hanging in the Senate Finance Committee, would provide income tax deferrals for self-employed persons setting aside money for private retirement plans.

Program and Progress

A National Republican Committee on *Program and Progress* proposed a far-reaching health program to be carried out by the Federal government in partnership with states and local governments.

Its goals would include: enlarging the capacity of medical schools so that 3,000 more doctors could be graduated each year, providing more hospital and nursing home beds, and supplementing hospital facilities with clinics, day-care centers, and more visiting nurses to care for patients in their own homes.

The progress of medical science would be furthered by continued Federal support for basic medical research. But such Federal support would be given under conditions to encourage maximum non-Federal spending on medical research and to prevent "too great a diversion . . . of doctors required for the equally urgent needs of teaching and medical practice." It was estimated that expenditure of $1 billion a year—equally divided between the Federal Government and non-Federal sources—would be required by 1965.

Other recommendations included: vigorous Federal

(Continued on page 525)

The Kansas Press Looks at Medicine

Editor's Note. In this section the JOURNAL reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.

UNIONS WANT OWN MEDICAL SYSTEM

Pushing their fight against the American Medical Association and the hospitals which they struck last summer, labor unions of New York State have plans to build their own hospital chain and operate their own medical insurance system in competition with Blue Cross and Blue Shield. They even talk of establishing medical schools to train physicians.

Whether anything ever comes of it is to be seen. It may be similar to union leader complaints about press and the talk of establishing newspapers. But they never get around to it.

But New York labor leaders have strong feelings about the hospitals, which they say have "sweatshop health standards," and about the medical insurance system. Big business, it is contended, dominates the insurance system and labor is outvoted. They want a system of benefit to the people.

That the unions have tremendous resources for these projects is true. The 26 New York unions interested in the proposal have combined reserves of $750 million in pension-welfare funds jointly administered by labor and management. Whether the unions could get employers and neutral trustees to agree to the ambitious program is doubtful. It would be risky to apply the funds to insurance programs, hospitals and medical schools that might show losses.

If this project it doubtful, however, the backing of unions for legislation in the next session of Congress to win more liberal medical treatment for aging and other citizens is certain.—*Wichita Eagle*, September 26, 1959.

A HITCH IN THE BELT

You can discuss the current local county-versus-medical vendors argument on two levels. There is the practical, factual dispute that involves percentages, per diem, prescription price lists, and contracts. That is the level on which the argument has been discussed for a long time.

While we like to think we're generally pretty practical in our approach, we much prefer to discuss the welfare medical issue on its theoretical, philosophical level. Don't run. Actually, the layman can make a great deal more sense out of the philosophical issue here than the practical level, which is overburdened with figures nobody can prove.

Try these basic questions on for size:

1. How much medical care do we want welfare patients to receive?

To help you ponder this issue remember that "the best" these days usually means expensive antibiotics, a hospital stay, tests and all of the latest developments known to medical science. When you contract the flu, you yourself must decide, "Do I call the doctor for treatment and a prescription, or do I just ride it out until the bugs get tired and quit?" "The best" would involve calling the doctor and possibly going to the hospital.

There are levels of medical care, and to a certain extent we determine the level we want by how much we want to spend. If the welfare client doesn't personally pay the medical bill (which he doesn't), then he naturally asks for "the best." Barton County's doctors indicate that the county's welfare clients have been getting the best care available. They say the welfare department has on occasion encouraged them to give less than the best. What does the public want in the way of medical care for welfare patients?

2. Assuming you have set the level of care that should be made available to the welfare client, who should bear the burden of the costs incurred?

In answering this question, you should bear in mind that there was a time when there were few expensive drugs, few hospital beds. Doctors treated

welfare patients in their offices, or in the patient's home. It was relatively simple, and in those days, the doctors did much of the welfare work without charge, or at minor expense to the county. Bear in mind also that medical expenses now lean heavily toward the hospitals and drugs, where the vendors have fixed expenses. Remember that hospitals are tax-free, charitable institutions, but are also non-profit, depending on income from hospital charges to meet expenses.

3. Should the county welfare board negotiate with the medical vendors for rates under those paid by the general public?

Remember that the county takes bids or otherwise negotiates with vendors for cars, asphalt, pencils and most of the other things it buys. But remember also that the county does not ask grocers to sell food to welfare clients at prices less than those paid by the public. And more important, remember that medical vendors are dispensing professional talent and services in addition to products. One doesn't ordinarily seek bids on an appendectomy.

Barton County's dispute is far from unique. Many Kansas counties are in the throes of a welfare medical payment hassle. Some county governments pay much less per welfare client for medical care. Chances are in these counties, fewer welfare clients are sent to the hospital and medical care is not so advanced as it is here. Some counties pay more per patient than has been paid here (though not very many).

The variations have developed through the years, without the aid of legislation. As a result, neither the welfare boards nor the medical vendors can call upon state statutes to settle their differences. The medical vendors traditionally have provided services to all, and are charged with accepting patients in hospitals as charitable institutions. So, while the county has financial resources with which to pay for welfare clients' medical expenses, the county can refuse to pay the full bill and get away with it. The county can't be sued.

The wide variation in welfare medical costs across the state has prompted the State Board of Social Welfare to urge county boards to hold the line on medical payments. But they have done so without telling the counties what level of medical care the welfare patients should receive. Nor have they stated just where the burden of paying the costs should fall.

These questions will eventually have to be settled on a statewide basis. But we have no guarantee of when that much-needed action will come. In the meantime, we can't have this local quarrel going on and on without settlement.

That means that the Barton County public must indicate to its elected commissioners what the public wants. Every taxpayer contributes towards the welfare department's operation. The welfare program should be and would be responsive to a public expression on the basic issues involved. The *Tribune's* Public Forum is open to those who have opinions on this important local problem.—*Great Bend Daily Tribune,* September 20, 1959.

KU Medical Center Doing Research on Polio Pills

If you are among those people who would prefer a beating to a shot from a hypodermic needle, then you will be delighted to know that another vaccine is being developed in pill form.

The University of Kansas Medical Center has been chosen to do research and test anti-polio pills. The pills will be tested on humans before they are adopted for general use.

The *Wichita Eagle* said recently that Dr. Herbert A. Wenner, in charge of virus studies at the Center, reports that the pills—believed to give lifetime immunity against paralytic polio—will be tried first on a pilot group of 20 youngsters, and later on thousands of children.

The *Eagle* pointed out that this is of tremendous importance; for if the tests prove the pills, millions of persons can be protected permanently without the difficulty of getting repeated shots.—*Coffeyville Journal,* October 8, 1959.

Washington Highlights

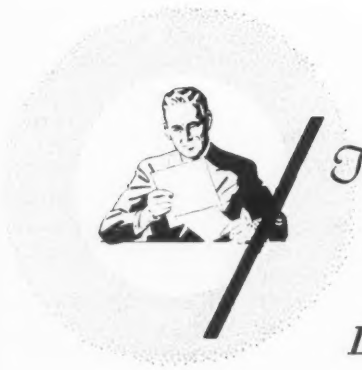
(Continued from page 523)

support of preventive health programs, and expansion and greater flexibility of voluntary health insurance programs.

"A free people and a free medical profession can achieve these goals with the wise support of government, without bureaucratic restrictions or interference with the physician-patient relationship which has made American health services a model for the free world," the Republican Committee stated.

Five-Point Plan

The Committee proposed a five-point *partnership* program: 1) short-term Federal aid for construction of medical school buildings, 2) changes in the present hospital construction program to encourage renovation and repair of outmoded hospitals, 3) Federal guarantees for mortgages to finance construction of private nursing homes on a basis assuring high standards of quality in construction and operation, 4) encouragement of construction of diagnostic and outpatient facilities in rural areas and the building of mental health clinics, and 5) Federal aid to cities "in more effective planning and coordination of health services."



The Business Side of Medicine

Liquidity Is Important to Your Estate

FLOYD F. WEHRENBERG, ***Kansas City, Missouri***

The meaning and importance of liquid assets are well known; that is, assets that can easily and quickly be converted to cash. By the same token, liquidity is just as important in the expedient settlement of an estate as in the operation of a business.

Without the necessary cash or liquid assets in an estate, there may be substantial losses and in some cases the entire estate plan will be placed in jeopardy. Because this subject is of primary importance to all who are systematically planning their estates, some of the pertinent problems of liquidity will be considered in this article.

Estimating the need for cash is often one of the most difficult things to do. The average person is seldom aware of the amount of cash that will be required to settle his estate. Medical, burial, and other last expenses as well as current unpaid bills, mortgages, and notes will all have to be settled. In addition there will be income tax on the final tax returns of the decedent in most cases, court costs, and administrative costs, and finally the federal estate and state inheritance taxes will fall due within a period of 15 to 18 months after death. It is obvious that in the case of a comparatively large estate, the amount of cash needed could amount to several thousands of dollars. If this is not available, substantial and unnecessary losses may result. Valuable property may have to be forfeited in an unfavorable market or a controlling business interest may have to be sacrificed. Accurate estimating of cash needs, then, is vital in planning for adequate liquidity and should be based on the preceding factors as precisely as possible.

The best type of liquid assets often poses a problem. Generally speaking, cash for the settlement of an estate should come from the following sources: cash, savings and checking accounts, corporate, municipal, and United States Government Bonds and marketable securities. At the time your estate plan is initiated, you should consult your attorney, your life insurance agent, business consultant, and broker before designating the assets to be used for cash needs.

Another factor to be considered is the *availability of the liquid assets to the estate.* In many estates there are sufficient liquid assets but they may not be available. For example, bank accounts and bonds are often held in joint tenancy with the right of survivorship. In such cases where these assets are intended to provide liquidity, they may instead pass directly to another person by operation of law rather than the will or estate plan.

What of a business interest? If the business is to be liquidated, the administrator or executor should have the right to operate it for a reasonable period of time in order to liquidate it under the most favorable circumstances. This will only be possible if the necessary funds are available to the administrator or executor. Liquidity will also be a controlling influence over the terms of a sale of the business and will govern whether or not a contract or mortgage sale can be made.

If the business is to be carried on, obviously sufficient available cash is essential to its efficient operation, and this cash should not have to be procured by forced sale of some other asset.

The problem of liquidity in the settlement of an estate is real and important. And since it is subject to constant change it should be reviewed and revised periodically by the property owner under the guidance of an experienced attorney and financial counselor. By following this policy, you will be able to realize your estate plan and provide maximum protection for your loved ones.

Mr. Wehrenberg is Missouri-Kansas manager, Professional Management Midwest, 4010 Washington Street, Kansas City, Missouri.

Blue Shield

Blue Cross-Blue Shield Rates Raised

New Blue Cross-Blue Shield rates for non-group and Farm Bureau members and certain employee groups have been submitted to the State Department of Insurance. Following approval by the Insurance Department these new rates will go into effect in January for most group members; in February for non-group members, and March for Farm Bureau members.

The proposed new rates are as follows, based on the $10 deductible for Blue Cross, and Plan A Blue Shield: (Blue Shield Plan B rates will be increased proportionately.)

Want All Members to Understand

Full information will be supplied to all Blue Cross-Blue Shield members before *any* rate changes take place. Upon approval by the Insurance Department, a letter of explanation from the presidents of the two Plans, accompanied by other basic information concerning the reasons and need for the increase will be mailed to all groups, and to non-group and Farm Bureau members preceding their billings in February and March.

In the meantime, meetings are being held with group, Farm Bureau and non-group members, explaining the need for an adjustment in dues. At the nine District Member Council meetings in October a complete report of the financial status of both Blue Cross and Blue Shield was heard, along with a discussion of recent trends in the utilization of services of both Plans. Members were given an opportunity to consider possible options, like deductibles, as a part of their present programs.

We repeat here some of the information which has been presented to members during the past several weeks:

- For the twelve month period ending July 1, 1959, each dollar of income was spent like this:

In Blue Cross:	95.4	cents spent for cases
	6.4	cents operating expenses
	101.8	is the total spent per dollar income
In Blue Shield:	94.1	cents spent for cases
	11.8	cents operating expenses
	105.9	cents is the total spent per dollar income

- Combined operating expense for the two Plans is only 8.2 cents per dollar.
- Last year it cost hospitals an average of $23.25 a day for patient care. This year, one day's hospital care is averaging about $25.00. Next year, hospital costs are expected to be approximately $26.85.
- Use of hospital services is still increasing. Last year there were 1,197 days of hospital care for each 1,000 members. This year it increased to about 1,230

(Continued on page 528)

	Present			*Proposed Rates*			*Percentage Increase*		
	BC	*BS*	*BC & BS*	*BC*	*BS*	*BC & BS*	*BC*	*BS*	*BC & BS*
Group Family	$ 6.80	$ 3.00	$ 9.80	$ 7.40	$ 3.70	$11.10	8.8	23.3	13.3
Non-Group Family	$ 8.10	$ 3.30	$11.40	$ 9.60	$ 4.50	$14.10	18.5	36.4	23.7
Farm Bureau Family	$ 7.30	$ 3.30	$10.60	$ 8.10	$ 4.40	$12.50	11.0	33.3	17.9

County SOCIETIES—CURRENT ACTIVITY

The **Cherokee County Medical Society** held a meeting at the Blue Castle Cafe in Baxter Springs recently.

Resolution was made to urge the Cherokee County Health Department to have a clinic for immunizations of polio, diphtheria, pertussis, tetanus, and small pox.

As a result of this meeting a clinic will be held in the county. The Society will also invite Dr. Paul Lawson of the South-East Kansas hospitals at Chanute, to conduct the tuberculin testing program in the county.

The **Golden Belt Medical Society** held a meeting last month at the Concordia Country Club in Concordia. Included in its program were the topics of "Management of Compression," "Fractures of the Dorsal and Lumbar Spine," by Dr. Cline Hensley, Wichita, and "Clinico-Pathological Conference," conducted by Clarence J. Weber, Salina. Refreshments and a dinner were served at the country club after which a business meeting was held.

"Your Heart" was the theme of a health meeting that was held in the city auditorium last month. It was sponsored by the **Shawnee County Medical Society** and The Kansas Heart Association and the Shawnee County Extension Service. Guest speakers were Dr. Grey Dimond of the University of Kansas Medical Center, Director of Cardiac Research. Films were also shown.

A panel of local doctors moderated by Dr. J. L. Lattimore answered questions. Other members of the panel were Dr. D. R. Bedford, Dr. John E. Crary, and Dwight Lawson.

Dr. Glenn R. Peters, president of the Kansas Medical Society, was speaker at a recent dinner meeting of the **Miami County Medical Society** and the Hospital Board of Trustees in the Miami County Hospital in Paola.

The doctors of the **Central Kansas Medical Society** met Thursday, September 17, at the country club at Ellsworth.

The scientific program was furnished by Dr. Wray Enders, anesthesiologist from St. Margaret's Hospital in Kansas City, Kansas. He spoke on his experiences with a recently developed anesthetic agent. Dr. Robert E. Allen, an oral surgeon from Kansas City, spoke on the subject of the immediate treatment of crushing face injuries.

Blue Shield

(Continued from page 527)

days. In 1960, it will be 1,265 days if the present upward trend continues.

- The use of physicians' services covered by Blue Shield is continuing to increase at the rate of 5 per cent a year.
- Whenever the cost or use of hospital-medical care increases, Blue Cross-Blue Shield pays more for services received. For example, if the cost of a semi-private room, drugs, dressings, laboratory, and oxygen increases 7 per cent, Blue Cross will be paying out 7 per cent more in 1960.
- The cost per member to administer Blue Cross-Blue Shield has remained fairly constant the last several years. As a per cent of income, operating costs have decreased from 9 per cent in 1958 to approximately 8.2 per cent in 1959.

The new interest rates on Series E and H Savings Bonds improve the yield on old bonds as well as on "new" ones bought since June 1, 1959.

KMS Obituaries

MILDRED CURTIS, M.D.

Dr. Mildred Curtis, 79, Iola physician, died October 4, at the Mercy Hospital, Fort Scott. She had been in failing health for some time.

Doctor Curtis was born in Geneva, grew to womanhood there and attended the University of Kansas Medical School. After receiving her diploma she returned to Iola in 1906 and practiced medicine there until a short time before her death.

She is survived by her half brother, two stepbrothers, and a stepsister.

Doctor Curtis was a member of the Allen County Medical Society since 1953.

FRED E. HARVEY, M.D.

Dr. Fred E. Harvey, 77, prominent Ottawa County physician, died the morning of August 6 at a Concordia rest home.

Born in 1881 in Ottawa County, Doctor Harvey had practiced medicine in Minneapolis from 1905 until the time of his retirement in 1954.

He served several terms as Ottawa County Health Officer. Doctor Harvey worked in field hospitals in France as an Army Major in World War I and later served with the American Army of Occupation in Germany.

He was a member of the staffs of the Ashbury and St. John's Hospitals, Salina, and a member of the American Medical Association and the Golden Belt Medical Association.

A charter member of the Minneapolis Post of American Legion, he also belonged to Veterans of Foreign Wars at Minneapolis. His survivors include his wife, a son, and his mother.

Doctor Harvey had been a member of this Society since 1935.

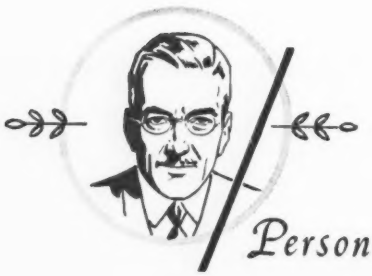
B. C. BEAL, M.D.

Dr. B. C. Beal, 83, practicing physician in Clearwater for more than 50 years, passed away on October 14. Dr. Beal died at St. Joseph Hospital. He was born June 18, 1876, in Tama, Iowa. He was a staff member of the St. Joseph Hospital and a member of county, state and national medical associations.

Dr. Beal was one of the oldest registered pharmacists in Kansas.

His survivors include his wife; a son, two daughters; brother, and three grandsons.

Doctor Beal was a member of the Sedgwick County Medical Society since 1933.



Personalities—IN KANSAS MEDICINE

Dr. C. D. Shrader has opened an office for the general practice of medicine and obstetrics in the United Building at Salina, Kansas.

Doctor Shrader has practiced in Newton for the past 12 years. He is a graduate of the University of Kansas School of Medicine in 1942, and served three years in the Navy.

Dr. A. E. Bair, Independence surgeon, recently returned home from an Atlantic City meeting where he was initiated as a fellow into the American College of Surgeons. He was one of the 1,013 surgeons from all over the world to be initiated.

A fellowship was also granted to **Dr. G. Bernard Joyce** of Topeka by the A.C.S.

A graduate of the University of Kansas School of Medicine in 1944, Doctor Joyce came to Topeka in 1953. He spent several years prior to that in the east studying orthopedics.

He is secretary of the executive medical staff of the Stormont-Vail Hospital, head of the Department of Surgery at St. Francis Hospital, and treasurer of the Shawnee County Medical Society.

Dr. Lynn O. Linton, Kansas City, and **Dr. Warren E. Meyer,** Wichita, also received a fellowship in the recent Atlantic City meeting of the American College of Surgeons. The A.C.S. founded in 1913 to establish standards of competency and character for specialists in surgery, has grown in 46 years' time from a founding group of 450 to a total membership of more than 23,250. Dr. Linton and Dr. Meyer were two of four Kansas physicians to receive the A.C.S. honor.

Dr. Karl Rottluff, Bonner Springs, announced last month that he is closing his office for private practice. Doctor Rottluff has been practicing medicine for more than 25 years.

Two Leavenworth physicians whose combined years of medical practice total more than a century, were honored by the Leavenworth County Medical Society recently. They are **Dr. D. R. Sterett** and **Dr. G. Ralph Combs.** They were cited at a dinner in their honor at the Hotel Cody in Leavenworth.

Doctor Sterett started practice in Saxman, Kansas in 1907, then moved to Winchester that year and to Leavenworth in 1916.

Doctor Combs began his medical career 57 years ago after he received his degree in medicine from the University of Pennsylvania.

The Hartig Clinic in Downs has a new associate, **Dr. Richard Boone.**

Doctor Boone graduated from the Kansas City College of Medicine and Surgery in 1920. He had a private practice and was a consultant and staff physician at Willow Maternity Hospital in Kansas City, Missouri for ten years. He had a general practice in White City, Kansas for 12 years also.

Doctor Boone also has had training in neuropsychiatry and geriatrics, and has been certified in geriatrics. Doctor Boone is a member of the Sedgwick County Medical Society, Wichita.

Dr. Larry E. Vin Zant, Wichita, was re-elected recently to the position of president of the Kansas Division of American Cancer Society. His election was part of the recent annual meeting of that society

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Margaret B. Obenschain, M.D.
First National Bank Bldg.
Winfield, Kansas

Lew W. Purinton, M.D.
839 S. Hillside
Wichita, Kansas

in which two projects came forth—the first being a group of 15,000 families in the state will be asked to take part in a six-year study to attempt to determine cases of cancer. The second project is the establishment of new "Living Research Memorials." Under this program all money donated as memorials will be used for research products.

Dr. A. H. Baum, Dodge City, was recently a speaker for the Achievement Day for the Ford County Home Demonstration units. His subject was "Use of Hypnosis in Medicine."

A recent honor was given to **Dr. Leroy A. Calkins,** Kansas City, when he was honored at a dinner given at Wolferman's in Kansas City, Missouri, by former resident physicians at the University of Kansas Medical Center, whose preceptor he was while they trained for their career for Ob-Gyn.

He was presented a portrait of himself done by Daniel MacMorris, a Kansas City portrait artist. It will hang in the Clendening Medical Library at the Medical Center along with portraits of other illustrious staff members, both living and deceased.

The presentation was made by Dr. T. J. Sims, first resident physician in Ob-Gyn at the Medical Center, and also the first under Dr. Calkins.

Doctor Calkins has recently passed his 65th birthday. He is a member of the Wyandotte County Medical Society.

C. P. C.

(Continued from page 518)

acute and chronic inflammatory cells in portal areas was present, and was probably related to the pancreatitis.

There was a striking degree of hemosiderosis of the spleen which weighed 250 gm. (normal 150 gm.). In addition, there were numerous acute focal necroses in the spleen which were part of the acute splenic enlargement, and were considered to be the result of the acute and chronic pancreatitis and the acute pericholangiohepatitis. An interesting fact is that terminally the bilirubin was slightly elevated and the hemoglobin had fallen to 10 gm. One could postulate that the patient was in the early stages of an acquired type of hemolytic anemia secondary to hypersplenism resulting from the acute splenic hyperplasia.

Grossly, esophageal varices were found, and microscopically extensive acute and chronic esophagitis with erosion of the mucosa compounds the problem of the varices. Gall described 20 cases of esophageal varices occurring in the absence of cirrhosis and associated with primary and metastatic cancer in the liver and even congestive heart failure. The varices and esophagitis were associated with partially digested blood in the stomach.

Dr. Delp: Thank you. Dr. Friesen, may we have your comments, please?

Dr. Friesen: At operation we found large, matted lymph nodes around the porta hepatis, larger nodes than one ordinarily sees even in metastatic disease. They were soft and large and completely surrounded the portal vein, common duct, and hepatic artery. On cut section they looked like sarcoma.

Also, at operation I saw hepatic metastases, umbilicated nodules within the liver which in themselves are typical of metastases, but they were atypical in that there was a stalk from each of these metastases of tissue growing to the parietal wall. They looked exactly like an inverted atom bomb. I believe that the shift of weight or of position and the weight of this liver on the parietal wall through these stalks probably produced much of the patient's pain on change of motion or of position. I had never seen such stalks before.

Dr. Delp: Dr. Klotz?

Dr. Klotz: A secretion test was attempted on the patient, but he was unable to swallow the tube. Although his temperature was normal, the mass was still as exquisitely tender as it was on admission. The initial gall bladder x-ray showed non-visualization of the gall bladder, and the follow-up intravenous iodipamide methylglucamine (Cholografin) was as Dr. Youngstrom described. My diagnosis was pericholecystic abscess in spite of the x-rays. The iodipamide methylglucamine may not have demonstrated stones that might have produced an inflammatory reaction.

Dr. Delp: A patient's presenting symptoms of fever and a painful mass in the upper abdomen does not immediately suggest carcinoma of the pancreas. Our patient died of erosive lesions in the upper part of the stomach, and I believe that is why he was bleeding. He did not have cirrhosis of the liver, but he did have varicosities. I am certain that the explanation for the varicosities are just those given by Dr. Friesen. The patient did have portal hypertension. I believe the changes in the spleen suggesting fibrocongestive splenomegaly were probably associated with it.

Pathological Anatomical Diagnosis

Carcinoma of the tail of the pancreas with metastases to regional lymph nodes and lungs.
Chronic pancreatitis.
Acute pericholangiohepatitis.
Esophageal varices.
Acute esophagitis.

Reference

Garret, N., and Gall, E.; Esophageal varices without hepatic cirrhosis. AMA Arch. of Path., 55:196, 1953.

Book REVIEWS

Synopsis of Ear, Nose, and Throat Diseases. By Robert E. Ryan, William C. Thornell and Hans Von Leden. Price $6.75. 383 pp. The C. V. Mosby Company, St. Louis, Mo.

This is a concise and standard presentation of otorhinolaryngic pathology. Each section is preceded by a discussion of applied anatomy and physiology. In many instances, the differential diagnosis and treatment is in outline form, and therefore somewhat brief and dogmatic. This is the type of book that would make a quick and convenient reference for the general practitioner, or a text for the medical student. —*R. E. B.*

Maloy's Evaluation of the Pelvis in Obstetrics, 2nd Edition. By Charles M. Steer, M.D. Price $4.00. 131 pp. Illustrated. W. B. Saunders Company, Philadelphia, Pa.

Maloy's evaluation of the pelvis in obstetrics is easy to read and understand. It is also sufficiently short enough that one can read it in one sitting. Yet it gives one the essential facts on which one can adequately approach the problem of difficult labor and make the correct decision as to how the difficult labor should be handled. Those who practice obstetrics should be familiar with its contents.—*J. L. S.*

Surgery in World War II—Neurosurgery, Volume 1. Prepared and published under the direction of Major General S. B. Hays. Price $5.00. 488 pp. U. S Government Printing Office, 1958.

This is the first time a book on neurosurgical principles of this magnitude has been written that is based on statistical data and evaluated by the actual war experiences of twelve outstanding neurosurgeons. In fifteen chapters and over 400 pages of well illustrated material the book deals with all facets of both administrative and clinical care of head injuries. Though concerned primarily with missile injuries to the brain and cranium during World War II, the general principles of diagnosis, treatment, and after care of all head injuries can be admirably used in civilian neurosurgical practice.—*R. C. T.*

Trauma. By Harrison L. McLaughlin, M.D. Price $18.00. 784 pp. Illustrated. W. B. Saunders Company, Philadelphia, Pa.

This book tends to broaden the field of trauma under a single cover in that it includes injuries other than the usual fracture type monograph.

The beginning of the book deals with various phases of the physiology of trauma in its response of the body. This pertains not only to bone and cell repair, but to a systemic response to injury.

The general principles in treatment of trauma should be well digested with careful thought by all types of practitioners.

Trauma of the specific regions not only include the various types of treatment, but a short résumé of the surgical anatomy and complications. Uncommon injuries are also discussed as to the prevention of mismanagement.

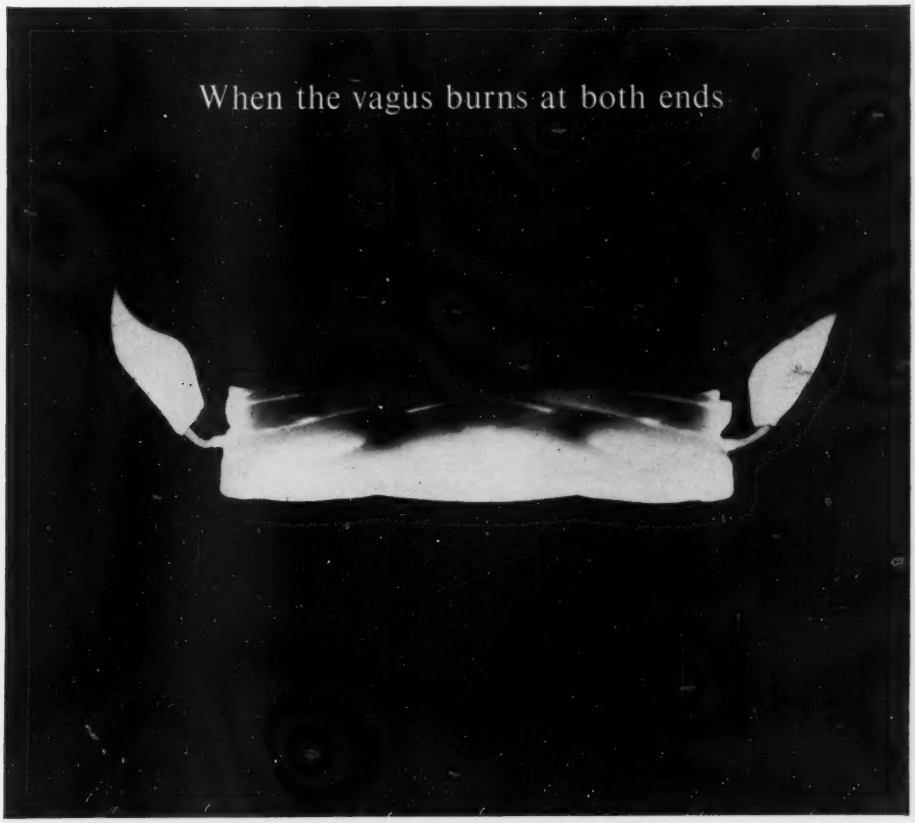
Many of the soft tissue injuries which are so often overlooked in fracture books are well handled as to recognition and treatment.

All fields of surgical specialties appear to be encompassed in each of their various types of injuries, but still preserving the relationship with each other for consideration of different approaches to the subject.

Fracture treatment discussions certainly do not present all of the forms of treatment, however, the presented procedures are well recognized as highly acceptable and efficient.

The procedures are not explained in a simple 1-2-3 manner, but with careful consideration as to some of the problems involved in reduction or with surgical intervention.

The completeness of this book without being voluminous makes it quite acceptable for those engaged in the treatment of trauma.—*G. B. J.*



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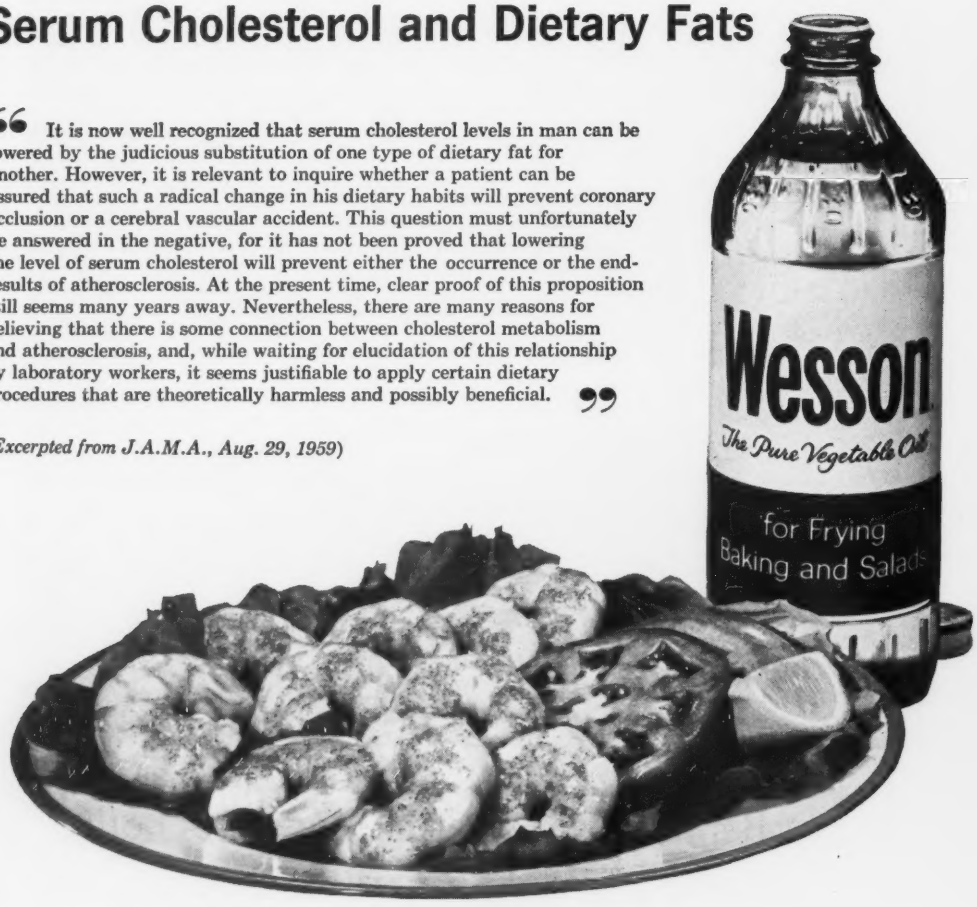
(*Excerpted from J.A.M.A., Aug. 29, 1959*)



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(1) Estimate based on epidemiologic study by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122, Jan. 1933.

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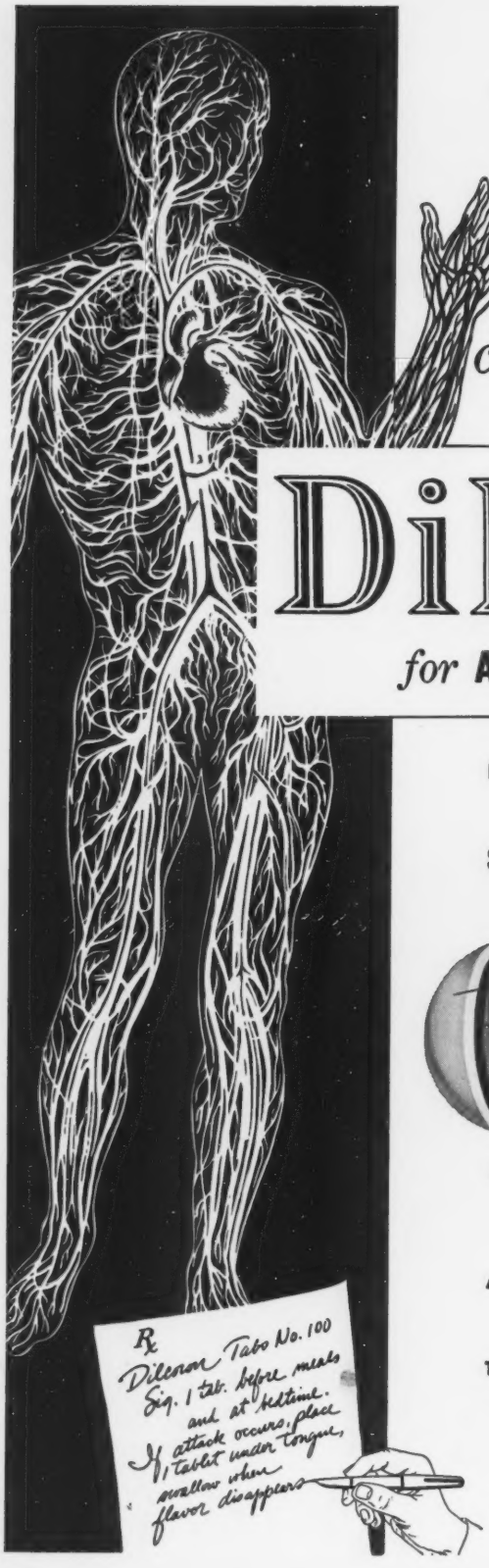

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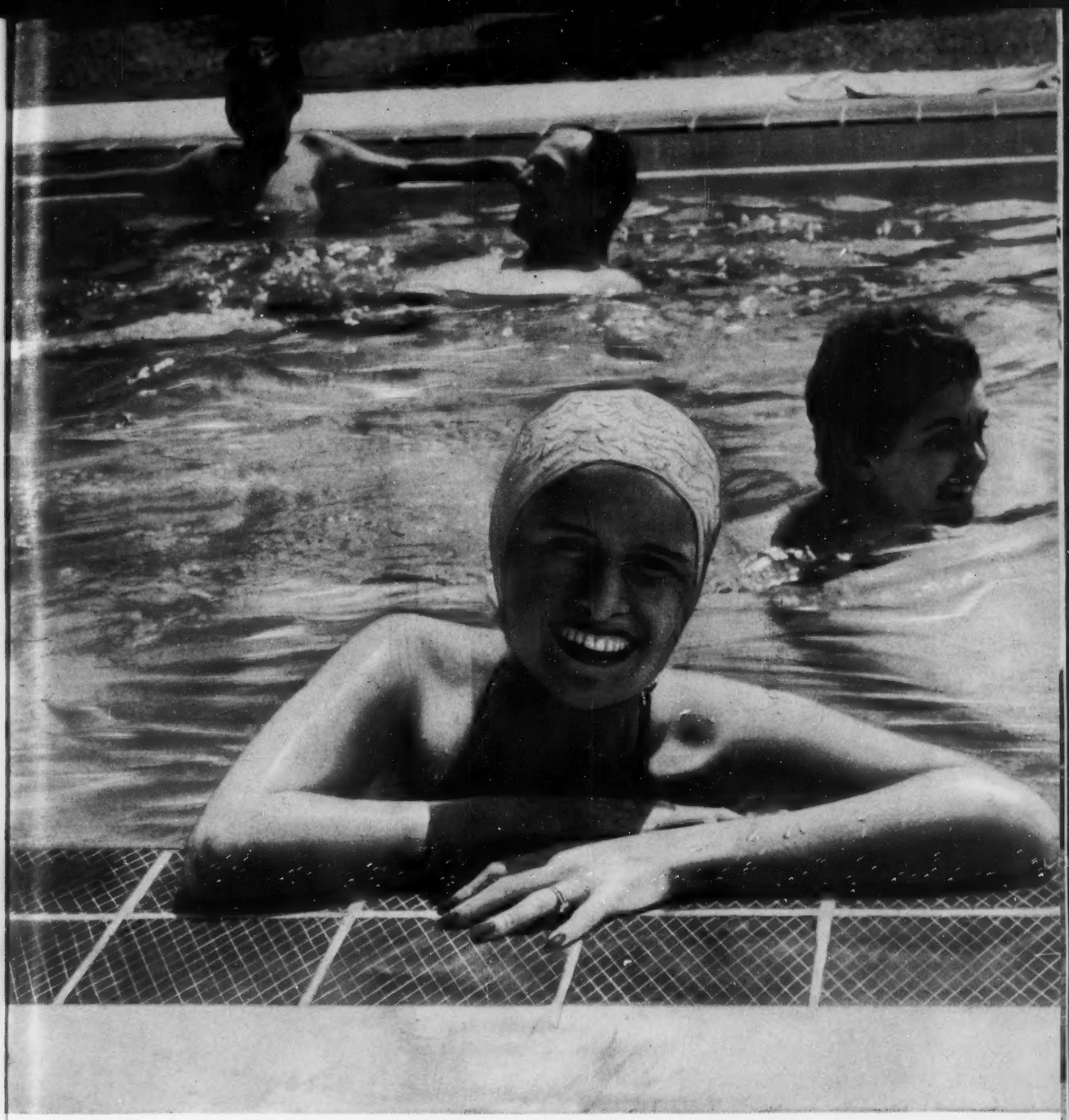

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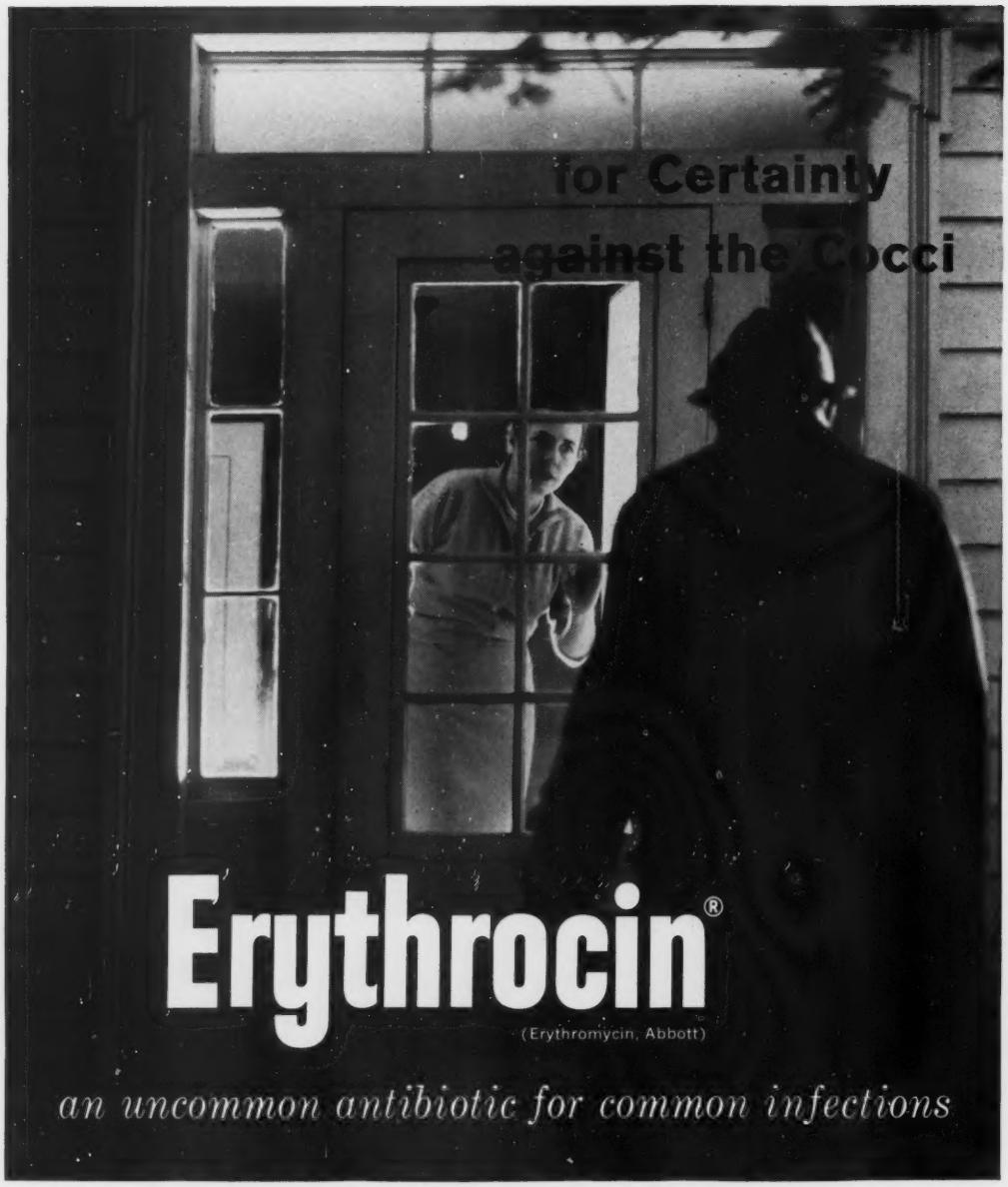

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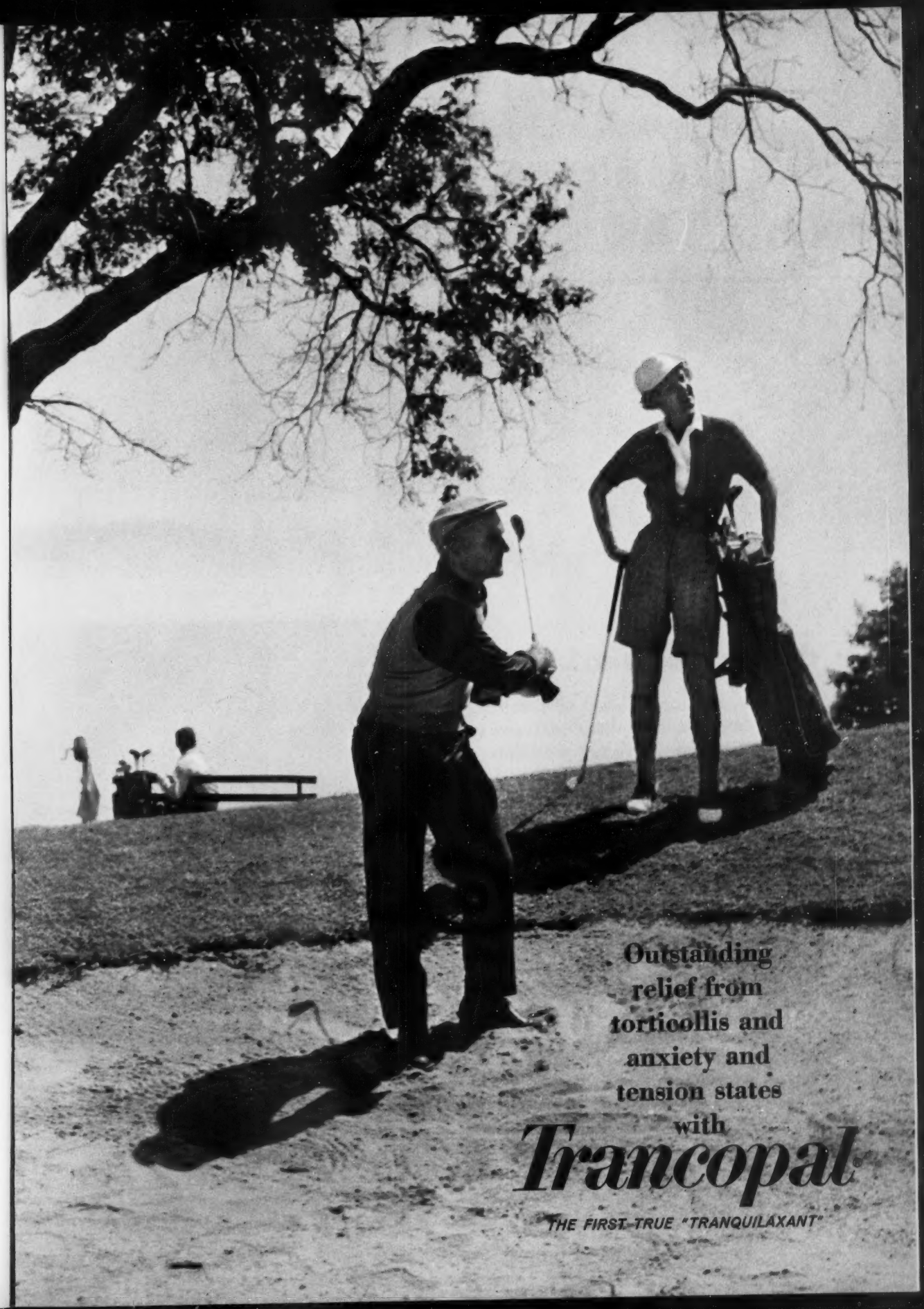
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anxiety and
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Trancopal
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Here is what you can expect when you prescribe

case profile no. 2840*

A 55-year-old man complained of a painful, very stiff neck on the left side. There was marked muscle spasm that seemed to involve primarily the trapezius muscle. He had a severe headache, with the pain radiating down the left side of the neck to the shoulder. There were no other findings on physical examination and results of routine laboratory tests were normal.

Trancopal was prescribed in a dosage of 200 mg. q.i.d. The first and second dose of Trancopal gave only moderate relief. However, after the third dose, there was marked relief of the stiffness of the neck, as well as the headache and shoulder pain.

After the fourth dose, medication was gradually decreased and was discontinued on the sixth day. One week later, the patient had moderate recurrence of the torticollis, and Trancopal was again prescribed in doses of 200 mg. q.i.d. The patient obtained complete relief in one day and no further treatment was required.

for torticollis

THE FIRST TRUE "TRANQUILAXANT"

Trancopal®

for anxiety and tension states

case profile no. 3382*

A 35-year-old woman, a professional model, had an acute, severe attack of anxiety. She was irrational and unable to eat, and was very restless.

Initial medication consisted of aspirin with codeine and later meprobamate. Neither was effective, and the patient's condition became worse. She had to be hospitalized because of the marked anxiety. Trancopal was then prescribed in a dosage of 200 mg. q.i.d., in addition to bed rest.

After the second dose of 200 mg. of Trancopal, the patient became calm and rational, and was able to eat. The dosage of Trancopal was gradually reduced to 100 mg. q.i.d. on the fourth hospital day, after which the patient was discharged and was able to return to her normal occupation.

**Clinical Reports on file at the Department of Medical Research, Winthrop Laboratories.*

Turn page for complete listings of Indications and Dosage.

THE FIRST TRUE "TRANQUILAXANT"

Trancopal

potent **MUSCLE RELAXANT**
effective **TRANQUILIZER**

Indications:

Musculoskeletal[1]	Psychogenic[1]
Neck pain (torticollis, etc.)	Anxiety and tension states
Low back pain (lumbago, etc.)	Dysmenorrhea
Bursitis	Premenstrual tension
Rheumatoid arthritis	Asthma
Osteoarthritis	Angina pectoris
Disc syndrome	Alcoholism
Fibrositis	
Ankle sprain, tennis elbow, etc.	
Myositis	
Postoperative muscle spasm	

Dosage: Adults, 100 or 200 mg. orally three or four times daily. Relief of symptoms occurs in fifteen to thirty minutes and lasts from four to six hours. The higher dosage is recommended for the treatment of patients in the acute stages of painful musculospastic conditions, and anxiety and tension states. Children (5 to 12 yrs.), 50 mg. three or four times daily.

Supply:

Trancopal Caplets®
100 mg. (peach colored, scored), bottles of 100.

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"The effect of this preparation in these cases [skeletal muscle spasm] was excellent and prompt . . ."[3]

". . . Trancopal is a most valuable drug for relieving tension, apprehension and various psychogenic states."[4]

1. Collective Study, Department of Medical Research, Winthrop Laboratories.
2. Lichtman, A. L. (N.Y. Polyclinic M. Sch. & Hosp.): Kentucky Acad. Gen. Pract. J. 4:28, Oct., 1958.
3. Mullin, W. G., and Epifano, Leonard (Long Island College Hosp.): Am. Pract. & Digest Treat. To be published.
4. Ganz, S. E. (New York, N. Y.): J. Indiana M. A. 52:1134, July, 1959.

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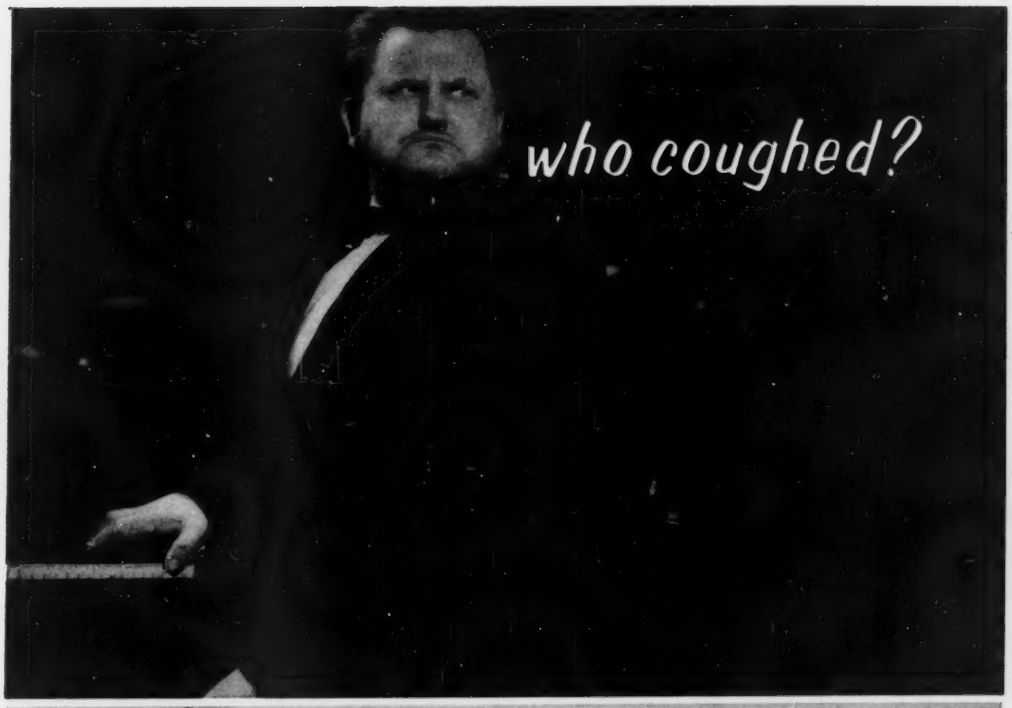

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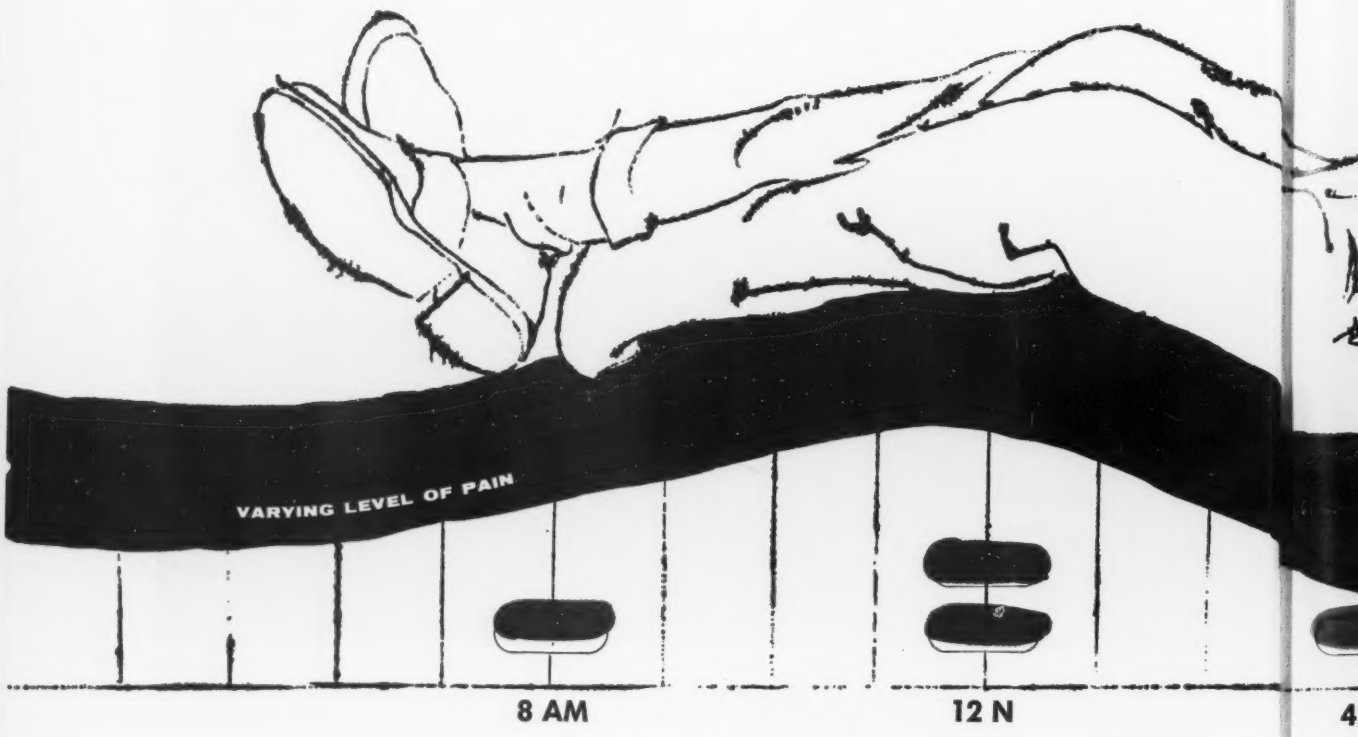

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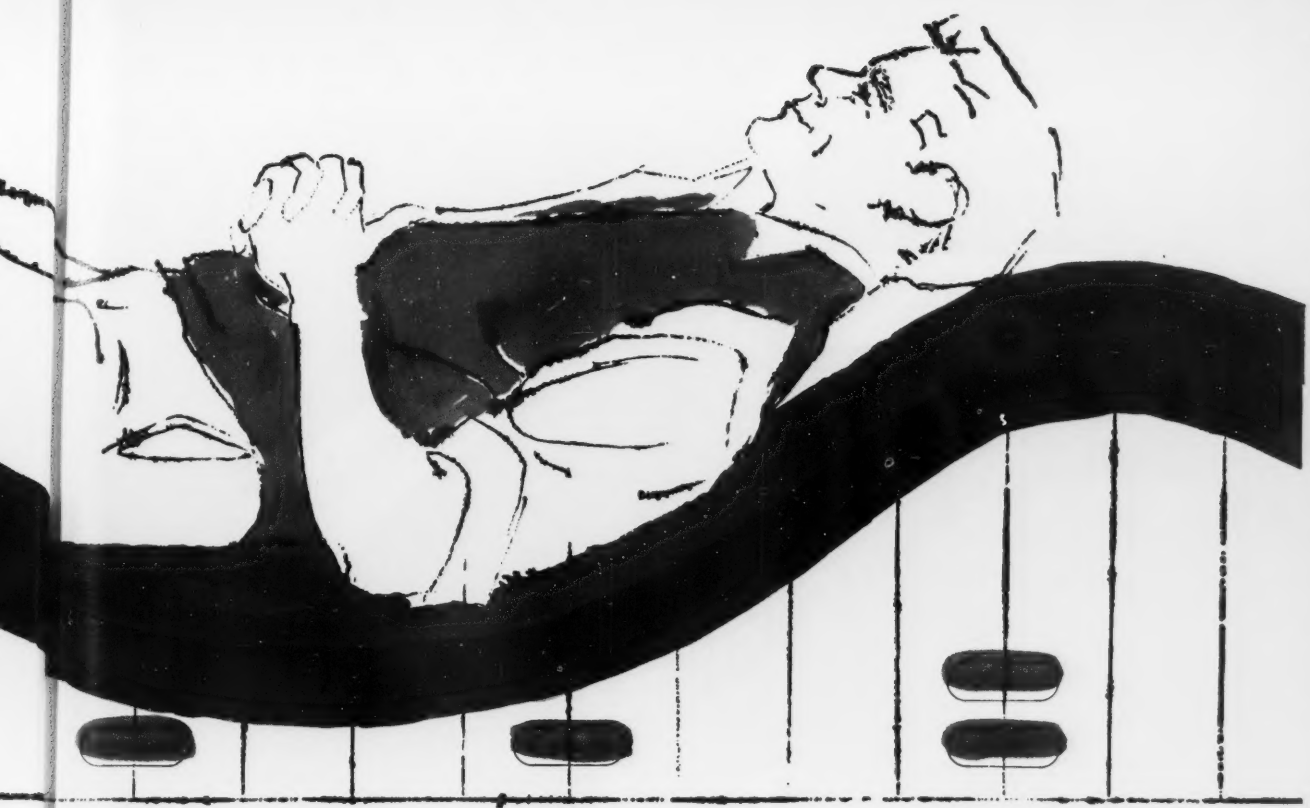

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The **right** dose of the **right** potency at the **right** time.

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For mild to moderate pain

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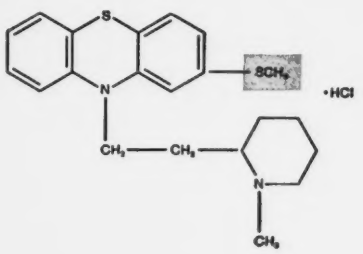
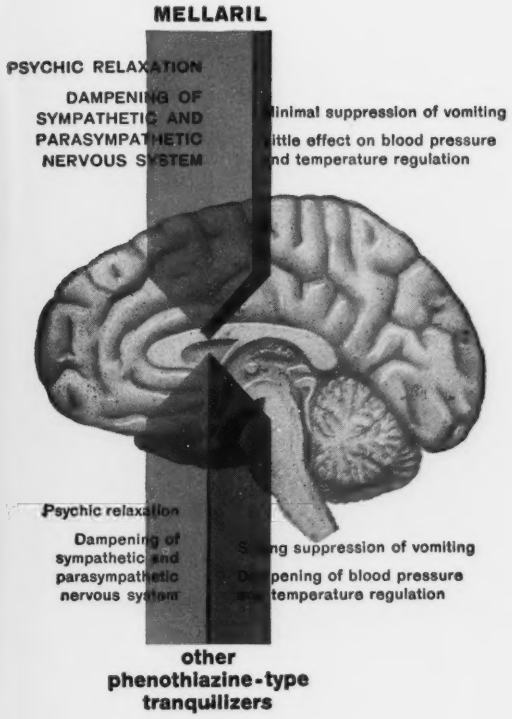
reater specificity of tranquilizing action results in fewer side effects



The presence of a thiomethyl radical (S-CH$_3$) is unique in Mellaril and could be responsible for the relative absence of side effects and greater specificity of psychotherapeutic action. This is shown clinically by:



1 A specificity of action on certain brain sites in contrast to the more generalized or "diffuse" action of other phenothiazines. This is evidenced by a lack of appreciable anti-emetic effect.

2 Less "spill-over" action to other brain areas — hence, absence of undue sedation, drowsiness or autonomic nervous system disturbances.

3 A notable absence of extrapyramidal stimulation.

4 Lack of impairment of patient's normal drive and energy.

5 Virtual freedom from such toxic effects as jaundice, photosensitivity, skin eruptions, blood forming disorders.

INDICATION	USUAL STARTING DOSE	TOTAL DAILY DOSAGE RANGE
ADULTS: Mental and Emotional Disturbances:		
MILD—where anxiety, apprehension and tension are present	10 mg. t.i.d.	20-60 mg.
MODERATE—where agitation exists in psychoneuroses, alcoholism, intractable pain, senility, etc.	25 mg. t.i.d.	50-200 mg.
SEVERE—in agitated psychotic states as schizophrenia, manic depressive, toxic psychoses, etc.:		
Ambulatory	100 mg. t.i.d.	200-400 mg.
Hospitalized	100 mg. t.i.d.	200-800 mg.
CHILDREN: BEHAVIOR PROBLEMS IN CHILDREN	10 mg. t.i.d.	20-40 mg.

MELLARIL Tablets, 10 mg., 25 mg., 100 mg.

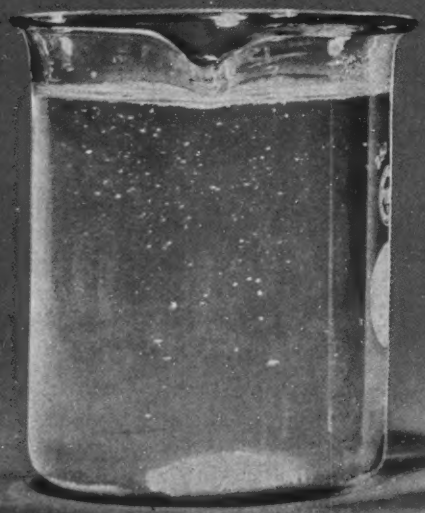
[a]Ostfeld, A. M.: Scientific Exhibit, American Academy of General Practice, San Francisco, April 6-9, 1959

SANDOZ

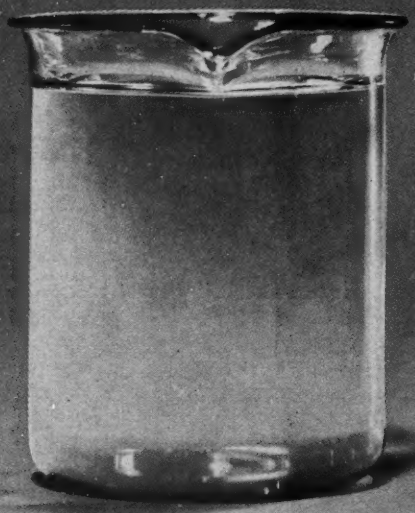
avoid the risk of insoluble, irritating aspirin particles

Chief among the drawbacks to aspirin usage is gastric intolerance. This ranges from mild upset and "heartburn" to severe hemorrhagic gastritis.[1-10] Studies performed in conjunction with gastrectomy[4, 5] and gastroscopy[2] have shown insoluble aspirin particles firmly adherent to the gastric mucosa and imbedded between rugae. Reactions varying from mild hyperemia to erosive gastritis have been reported to occur in the areas immediately surrounding these adherent particles.[2, 4, 5] This is reported to be particularly true in patients with peptic ulcer.[4]

CALURIN is the freely soluble, stable calcium aspirin complex. Its high solubility forestalls gastric irritation or damage



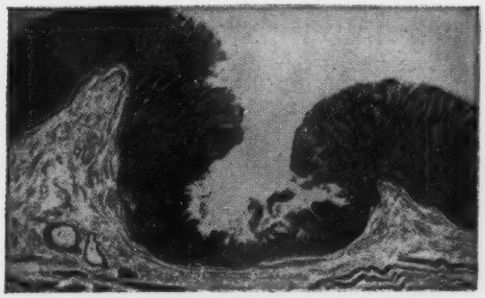
Regular aspirin crystals 24 hours after being mixed into water.



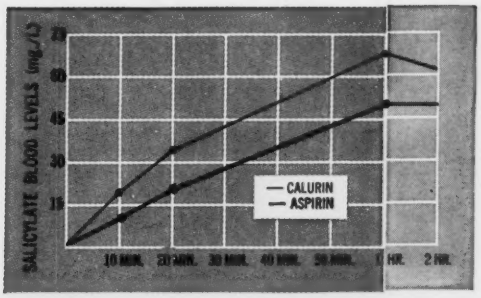
Calurin crystals in solution one minute after being mixed into water.

CALURIN*

STABLE SOLUBLE CALCIUM-ACETYLSALICYLATE-CARBAMIDE



Particle-induced ulceration — section through lesion found in gastrectomy specimen. An aspirin particle was found firmly imbedded in this undermined erosion. Such lesions may be associated with the relative insolubility of aspirin, which remains in particulate form after dispersion in gastric contents.



Calurin, being freely soluble, is promptly available for absorption into the systemic circulation. Salicylate blood levels in 12 subjects receiving both Calurin and plain aspirin were found to rise more than twice as high within ten minutes following Calurin. Also, these levels persisted higher for at least two hours.[11]

CALURIN is the aspirin of choice, especially when high-dosage, long-term therapy is indicated:

1. High solubility forestalls gastric irritation or damage. This advantage is of special importance in arthritis and other conditions requiring high-dosage, long-term therapy.
2. Produces high salicylate blood levels rapidly for prompt analgesic, anti-pyretic, anti-arthritic effect.
3. Sodium-free — for safer long-term therapy.
4. Flavored: can be chewed or dissolved in the mouth without water if desired — an advantage for patients requiring aspirin administration during the night and for pediatric patients.

Dosage: Each tablet of Calurin is equivalent to 300 mg. (5 gr.) of acetylsalicylic acid. For relief of pain and fever in adult patients, the usual dose of Calurin is 1 to 3 tablets every 4 hours, as needed; in arthritic states, 2 or 3 tablets 3 or 4 times daily; in rheumatic fever, 3 to 5 tablets 4 or 5 times daily. For children over 6 years, the usual dose is 1 tablet every 4 hours; for children 3 to 6 years, ½ tablet every 4 hours, as required. Not recommended for children under 3.

REFERENCES: 1. Waterson, A. P.: Aspirin and gastric haemorrhage, Brit. M. J. **2**:1531, 1955. 2. Douthwaite, A. H., and Lintott, G. A. M.: Gastroscopic observation of the effect of aspirin and certain other substances on the stomach, Lancet **2**:1222, 1938. 3. Editorial Comments: The effect of acetylsalicylic acid (aspirin) on the gastric mucosa, Canad. M. A. J. **80**:47, 1959. 4. Muir, A., and Cossar, I. A.: Aspirin and ulcer, Brit. M. J. **2**:7, 1955. 5. Muir, A., and Cossar, I. A.: Aspirin and gastric haemorrhage, Lancet **1**:539, 1959. 6. Schneider, E. M.: Aspirin as a gastric irritant, Gastroenterology **33**:616, 1957. 7. Bayles, T. B., and Tenckhoff, H.: Salicylate therapy in rheumatic diseases, Scientific Exhibit, Ann. Mtg. A. M. A., San Francisco, Calif., June, 1958. 8. Batterman, R. C.: Comparison of buffered and unbuffered acetylsalicylic acid, New Eng. J. M. **258**:213, 1958. 9. Cronk, G. A.: Laboratory and clinical studies with buffered and nonbuffered acetylsalicylic acid, New Eng. J. M. **258**:219, 1958. 10. Editorial: Aspirin plain and buffered, Brit. M. J. **1**:349, 1959. 11. Smith, P. K.: Plasma concentration of salicylate after the administration of acetylsalicylic acid or calcium acetylsalicylate to human subjects, Report submitted to Smith-Dorsey from Dept. of Pharmacology, Geo. Washington Univ. School of Medicine, Washington, D. C., Sept. 5, 1958.

*TRADEMARK

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska

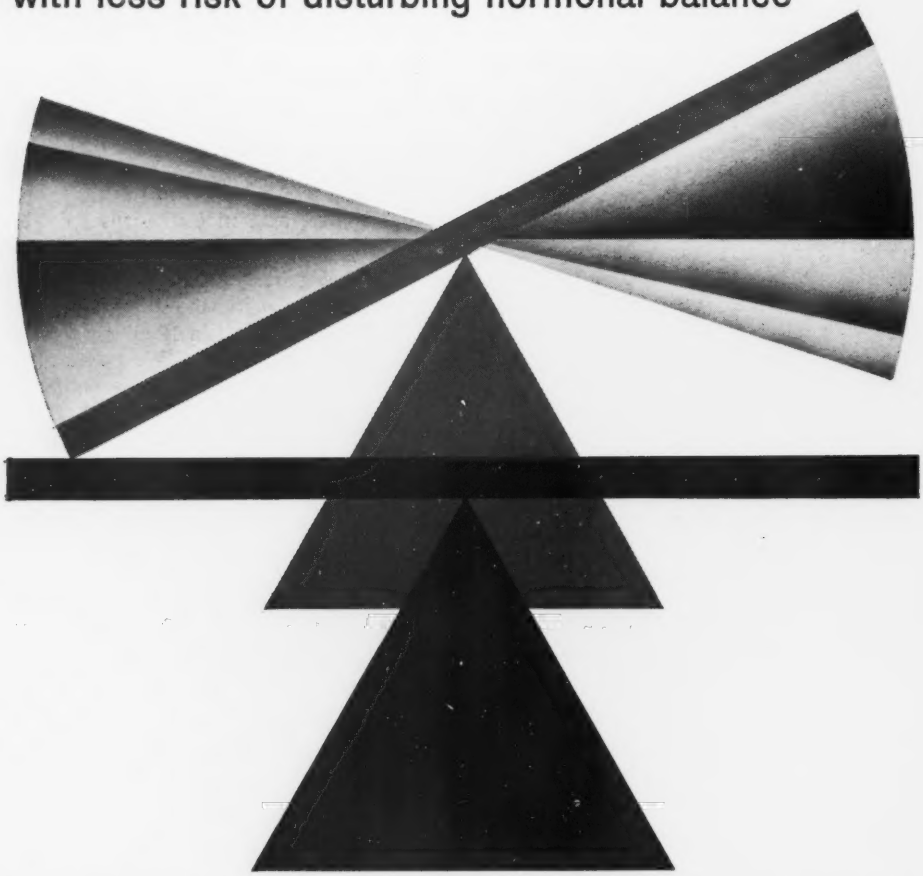
Effective relief in rheumatic disorders

Sterazolidin® capsules

prednisone-phenylbutazone Geigy

Geigy

with less risk of disturbing hormonal balance



In the treatment of the rheumatic disorders new Sterazolidin provides a method of limiting the gravest danger inherent in steroid therapy... hypercortisonism arising from excessive dosage.

Repeatedly it has been shown that the addition of low dosage of Butazolidin sharply reduces hormone requirement.[1-4] Sterazolidin is a combination of prednisone (1.25 mg.) and Butazolidin (50 mg.) which provides, in the majority of cases, consistent relief at a stable uniform maintenance dosage significantly below the level at which serious hormonal imbalance is likely to occur.

Sterazolidin® (prednisone-phenylbutazone Geigy). Each capsule contains prednisone 1.25 mg.; phenylbutazone 50 mg.; dried aluminum hydroxide gel 100 mg.; magnesium trisilicate 150 mg. and homatropine methylbromide 1.25 mg.

1. Kuzell, W. C., and others.: Arch. Int. Med. 92:646, 1953. 2. Wolfson, W. Q.: J. Michigan M. Soc. 54:323, 1955. 3. Strandberg, B.: Brit. J. Phys. Med. 19:9, 1956. 4. Platt, W. D., Jr., and Steinberg, I. H.: New England J. Med. 256:823 (May 2) 1957.

Geigy, Ardsley, New York

9880

NEO-SYNEP

CONTROL OF COMMON COLD SYMPTOMS with

Neo-Synephrine Compound Cold Tablets is pharmacologically comprehensive and clinically practical...

COMPREHENSIVE because in this new preparation are rationally combined drug actions that are needed to control the common cold symptomatology "across the board"

PRACTICAL because the average patient will promptly find relief from his distress.

EPHRINE® COLD TABLETS

COMPOUND

S

For full-range symptomatic relief orally each tablet of Neo-Synephrine Compound provides:

VS. NASAL STUFFINESS, TIGHTNESS—
Neo-Synephrine hydrochloride...5 mg.
pre-eminent, orally effective decongestant

VS. ACHES, CHILLS, FEVER—
Acetaminophen (N-acetyl-*p*-aminophenol)...150 mg.
modern analgesic and antipyretic

VS. RHINORRHEA—
Thenfadil® hydrochloride ...7.5 mg.
effective, well tolerated antihistaminic

VS. LASSITUDE, MALAISE—
Caffeine...15 mg.
dependable, mild, stimulating agent

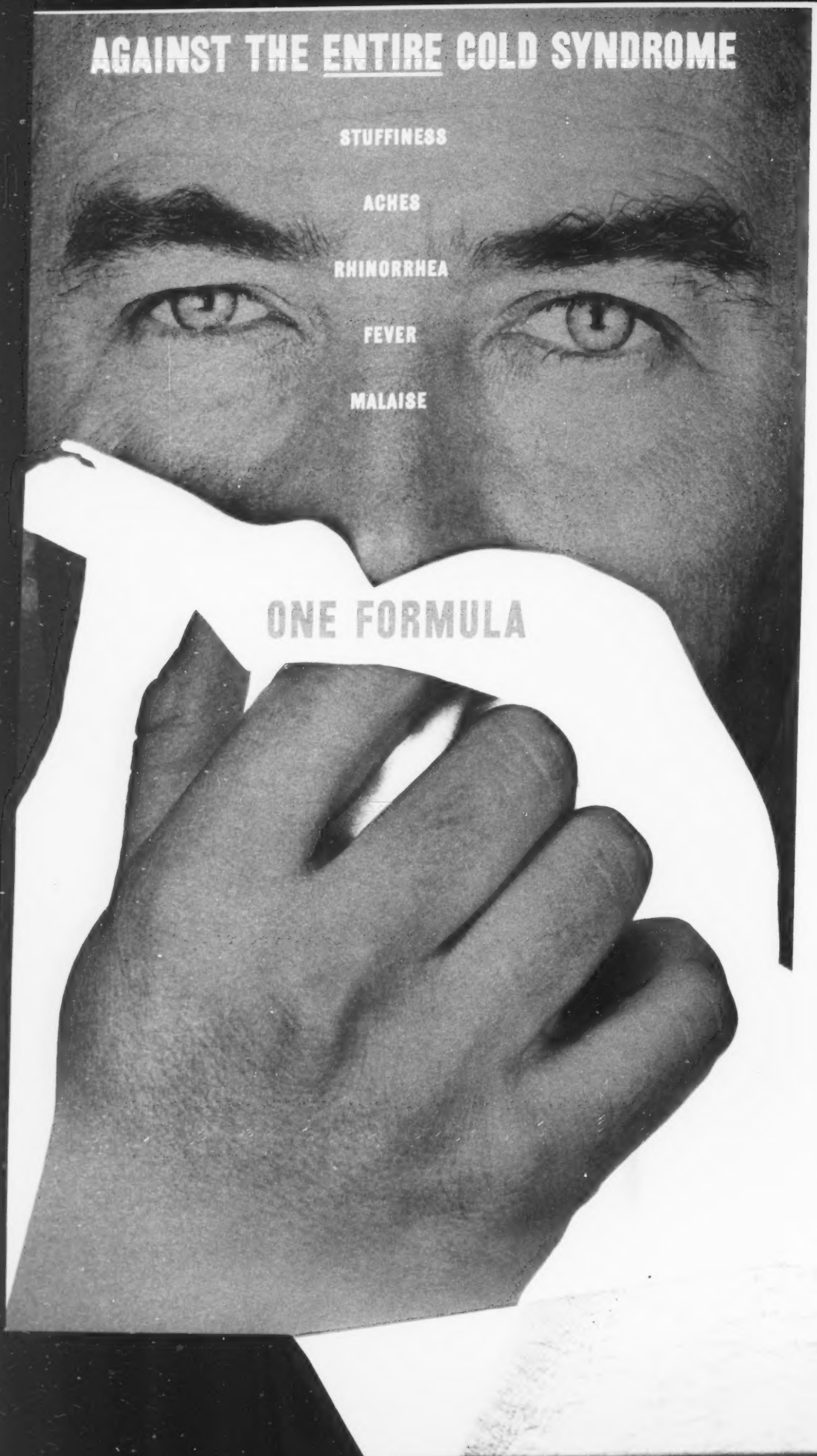
DOSAGE:

Adults—2 tablets three times a day

Children 6 to 12 years—
1 tablet three times a day

Bottles of 20 and 100 tablets

Winthrop LABORATORIES



quick
and
lasting
decongestion

Combining three effective intranasal medications, NTz produces sustained decongestion ...aeration...sinus drainage. There are virtually no side effects, and NTz maintains therapeutic action with repeated use. NTz is available in a convenient, nonbreakable plastic squeeze bottle of 20 cc. that delivers a fine, even spray and does not leak. Even small children use it easily.

Winthrop LABORATORIES

NTz. Neo-Syn
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TO CLEAR THE AIRWAY

topically in colds, sinusitis and allergic rhinitis

NTZ®

Nasal Spray

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***N**eo-Synephrine® hydrochloride, 0.5%* –
accepted vasoconstrictor and decongestant

***T**henfadil® hydrochloride, 0.1%* –
dependable topical antihistaminic

***Z**ephiran® chloride, 1:5000* –
antibacterial wetting agent and preservative

LABORATORIES *New York 18, N.Y.*

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Combining three effect
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...aeration...sinus dra
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produces sustained decongestion
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side effects, and NTZ maintains
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e in a convenient, nonbreakable
eeze bottle of 20 cc. that delivers
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ren use it easily.

***N**eo-Synephrine® hydrochloride, 0.5%* –
accepted vasoconstrictor and decongestant

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dependable topical antihistaminic

***Z**ephiran® chloride, 1:5000* –
antibacterial wetting agent and preservative

Winthrop LABORATORIES *New York 18, N.Y.*

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the disease of many masks

Doctor, do you recognize this patient? She complains of flatulence, constipation with alternating periods of diarrhea, and colicky pains in the lower right quadrant. At other times she is troubled by anorexia, lassitude, dull headache, muscle pains and backache. Or she may have only one or two of these symptoms.

In these puzzling cases, serious consideration should be given to intestinal amebiasis—the disease of many masks. Clinicians say it is "one of the most widespread and serious protozoan diseases of man," yet "there is no parasite more often misdiagnosed than is *E. histolytica*." Conservative estimates place the incidence at 10% of the United States population as a whole, and 16% in southern states.

Now Glarubin, a relatively non-toxic amebicide, simplifies the treatment of suspected cases of intestinal amebiasis. Glarubin, a crystalline glycoside from the fruit of *Simarouba glauca*, is a specific amebicidal agent with minimal side effects. It contains no arsenic, bismuth or iodine.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis, and virtually free of toxicity.

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin. Write for descriptive literature, bibliography, and dosage schedules.

new Glarubin

TABLETS

specific for intestinal amebiasis

THE S. E. MASSENGILL COMPANY

BRISTOL, TENNESSEE

NEW YORK • KANSAS CITY • SAN FRANCISCO

Blood pressure
before Apresoline-Esidrix:

206/118

mm.Hg*

Blood pressure after Apresoline-Esidrix:

182/98 mm. Hg*

Added benefits: Lowered dosage requirements, fewer side effects • Improved renal blood flow • Relaxed cerebral vascular tone • Excellent diuresis in decompensated cases

SUPPLIED: Apresoline-Esidrix Tablets (orange), each containing 25 mg. of Apresoline hydrochloride and 15 mg. of Esidrix; bottles of 100.

*Response of 56-year-old female patient noted in clinical report to CIBA.

APRESOLINE® hydrochloride (hydralazine hydrochloride CIBA) / ESIDRIX® (hydrochlorothiazide CIBA)

Apresoline®-Esidrix®

Combination Tablets

POTENTIATED ANTIHYPERTENSIVE FOR ADVANCING HYPERTENSION

CIBA

SUMMIT, NEW JERSEY

when the
rheumatic
disorder
is more
than salicylates
alone
can control...

MORE
HIGHLY INDIVIDUALIZED
THERAPY
FOR THE
RHEUMATIC
"IN-BETWEEN"

...but
control
requires less
than
intensive
steroid therapy
alone

Aristo

wider latitude in adjusting dosage

ARISTOGESIC is particularly effective for relief of chronic — but less severe — pain of rheumatic origin. ARISTOGESIC combines the anti-inflammatory effects of ARISTOCORT® Triamcinolone with the analgesic action of salicylamide, a highly potent salicylate. Dosage requirements for ARISTOGESIC are substantially lower than generally required for each agent alone. The exceptionally wide latitude of dosage adjustment with ARISTOGESIC permits well-tolerated therapy for long periods of time with fewer side effects.

Indications: Mild cases of rheumatoid arthritis, tenosynovitis, synovitis, bursitis, mild spondylitis, myositis, fibrositis, neuritis, and certain muscular strains.

Dosage: Average initial dosage: 2 capsules 3 or 4 times daily. Maintenance dosage to be adjusted according to response.

Precautions: All precautions and contraindications traditional to corticosteroid therapy should be observed. The amount of drug used should be carefully adjusted to the lowest dosage which will suppress symptoms. Discontinuance of therapy must be carried out gradually after patients have been on steroids for prolonged periods.

Each ARISTOGESIC Capsule contains:

ARISTOCORT® Triamcinolone	0.5 mg.
Salicylamide	325 mg.
Dried Aluminum Hydroxide Gel	75 mg.
Ascorbic Acid	20 mg.

Supply: Bottles of 100 and 1,000.

gesic® Capsules

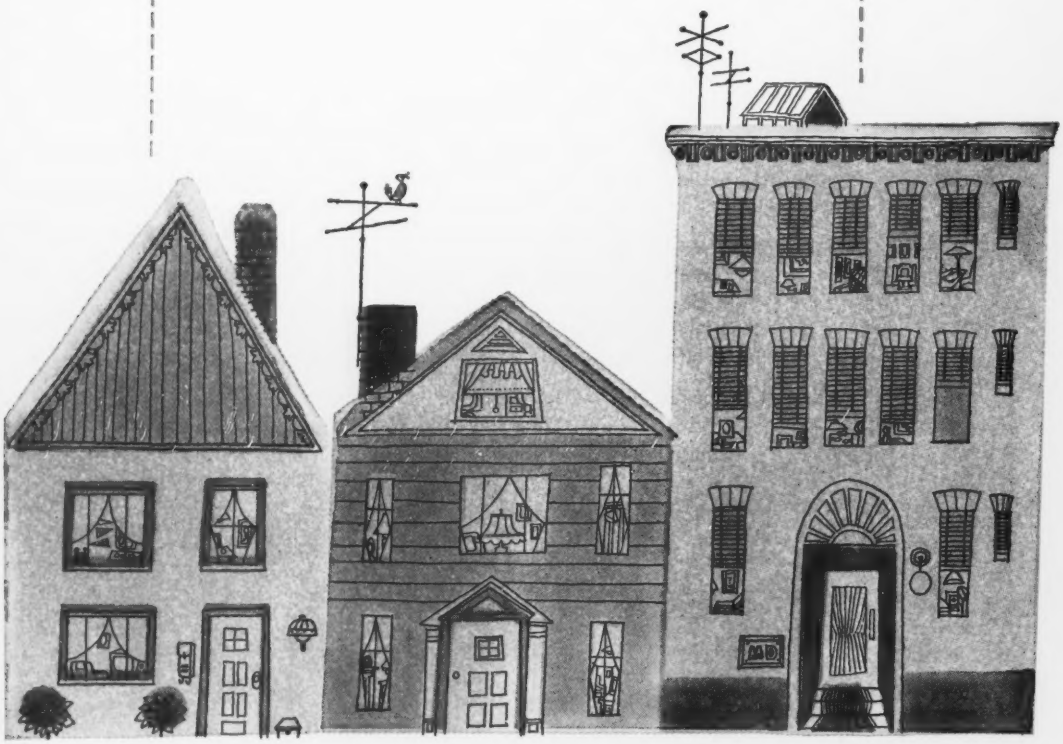
Steroid-Analgesic Compound LEDERLE

Lederle

LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

"But, Doctor, I just can't swallow a lot of tablets"

"Little mother, just
ONE
BONADOXIN®
tablet stops morning sickness (you take it at bedtime)"



The formula tells why BONADOXIN quickly stops nausea and vomiting of pregnancy in 9 out of 10 cases.* Each tiny BONADOXIN tablet contains:
Meclizine HCl (25 mg.) for antinauseant action / Pyridoxine HCl (50 mg.) for metabolic replacement

More than 60,000,000 tablets prescribed and taken. Toxicity low, tolerance excellent. In bottles of 25 and 100. Usual dose: one tablet at bedtime; severe cases may require another on arising. See PDR, p. 779.

BONADOXIN also effectively relieves nausea and vomiting associated with: anesthesia, radiation sickness, Meniere's syndrome, labyrinthitis, cerebral arteriosclerosis and motion sickness.

After Baby Comes

For infant colic, try antispasmodic BONADOXIN Drops... stop colic in 7 out of 8 cases.*

Each cc. contains:
Meclizine 8.33 mg. / Pyridoxine 16.67 mg.

See PDR, p. 779.

*Bibliography available on request.

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For the first time

CONVENIENCE and ECONOMY

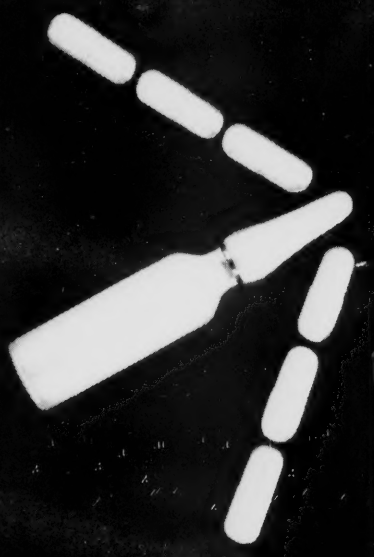
for that all-important first dose of broad-spectrum antibiotic therapy

New

TERRAMYCIN®

brand of oxytetracycline

INTRAMUSCULAR SOLUTION



Initiation of therapy in *minutes after diagnosis* with new, ready-to-inject Terramycin Intramuscular Solution provides maximum, sustained absorption of potent broad-spectrum activity.

...and for continued, compatible, coordinated therapy

COSA-TERRAMYCIN®

oxytetracycline with glucosamine

CAPSULES

Continuation with oral Cosa-Terramycin every six hours will provide highly effective antibacterial serum and tissue levels for prompt infection control.

The unsurpassed record of clinical effectiveness and safety established for Terramycin is your guide to successful antibiotic therapy.

Supply:

*Terramycin Intramuscular Solution**
100 mg./2 cc. ampules
250 mg./2 cc. ampules

Cosa-Terramycin Capsules
125 mg. and 250 mg.

Cosa-Terramycin is also available as:
Cosa-Terramycin Oral Suspension — peach flavored, 125 mg./5 cc., 2 oz. bottle
Cosa-Terramycin Pediatric Drops — peach flavored, 5 mg./drop (100 mg./cc.), 10 cc. bottle with plastic calibrated dropper

Complete information on Terramycin Intramuscular Solution and Cosa-Terramycin oral forms is available through your Pfizer Representative or the Medical Department, Pfizer Laboratories.

*Contains 2% Xylocaine® (lidocaine), trademark of Astra Pharmaceutical Products, Inc.

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PFIZER LABORATORIES, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N. Y.

Now —All cold symptoms can be controlled

Tussagesic

timed-release tablets

Controls congestion
with Triaminic,[1,2,3] the leading oral nasal decongestant.

Controls aches and fever
with well-tolerated APAP, non-addictive analgetic[4] and excellent antipyretic.[5]

Each TUSSAGESIC Tablet provides:

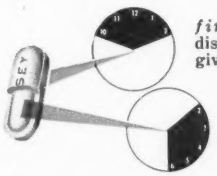
TRIAMINIC®	50 mg.
(phenylpropanolamine HCl	25 mg.
pheniramine maleate	12.5 mg.
pyrilamine maleate	12.5 mg.)
Dormethan (brand of dextromethorphan HBr)	30 mg.
Terpin hydrate	180 mg.
APAP (N-acetyl-p-aminophenol)	325 mg.

References: 1. Lhotka, F. M.: Illinois M. J. 112:259 (Dec.) 1957. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958. 4. Bonica, J. J.: in Drugs of Choice, Mosby, St. Louis, 1958, p. 272. 5. Dascomb, H. E.: in Current Therapy, Saunders, Phila., 1958, p.78. 6. Bickerman, H. A.: in Drugs of Choice, Mosby, St. Louis, 1958, p.547.

Controls cough centrally
with non-narcotic Dormethan, possessing "amply demonstrated" antitussive activity,[6] as effective as codeine.

Liquefies tenacious mucus
with terpin hydrate, classic expectorant.

Prompt and prolonged relief because of this special "timed release" design:



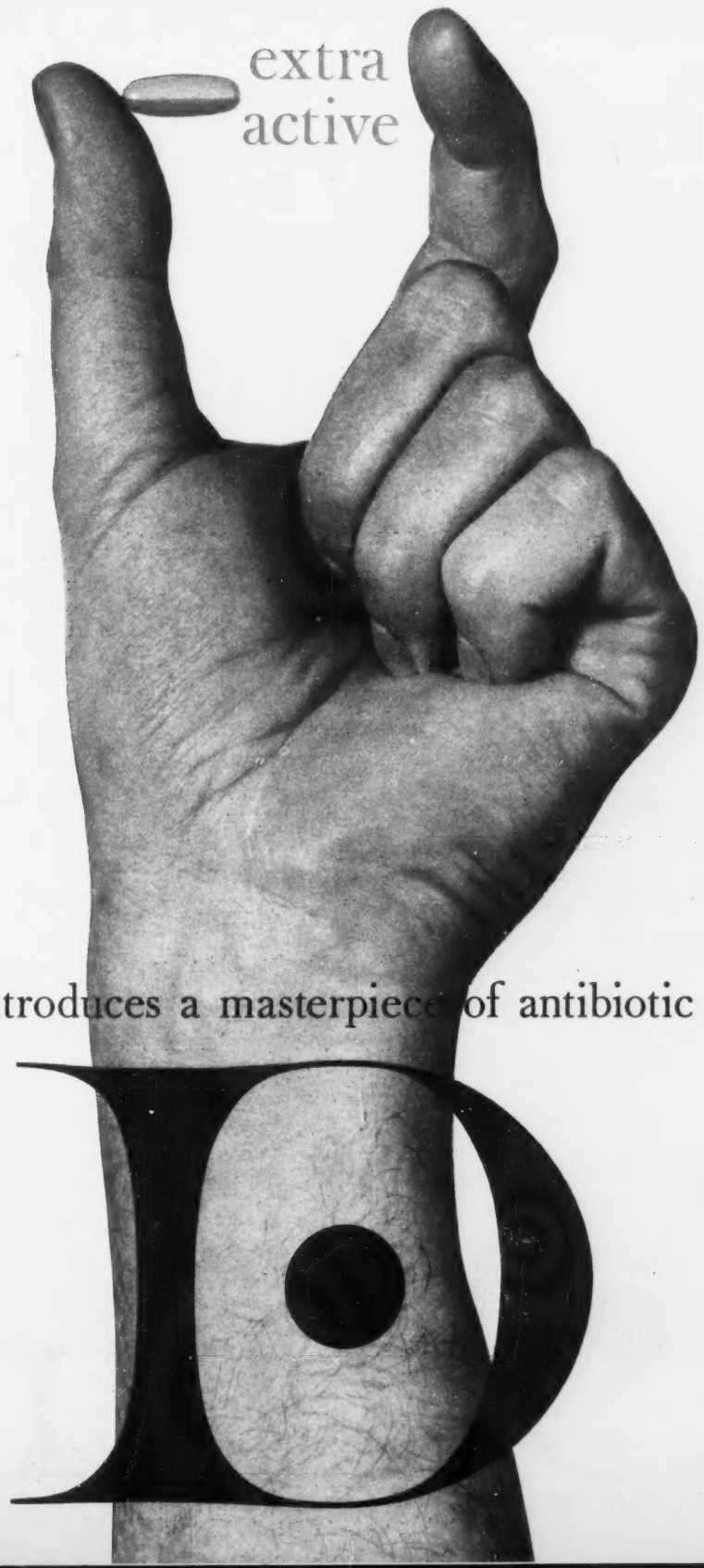
first — the outer layer dissolves within minutes to give 3 to 4 hours of relief

then — the inner core releases its ingredients to sustain relief for 3 to 4 more hours

Dosage: One tablet in the morning, midafternoon and at bedtime. Pediatric dosage chart for Tussagesic Suspension available on request.

TUSSAGESIC SUSPENSION provides palatability and convenience which make it especially attractive to children and other patients who prefer liquid medication.

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska



Lederle introduces a masterpiece of antibiotic design

Strikingly enhances the traditional advantages of broad-spectrum antibiotics...

for greater patient-physician benefit

DECLOMYCIN is a unique fermentation product of a strain of *Streptomyces aureofaciens*—the parent organism of AUREOMYCIN®* and ACHROMYCIN.®†

DECLOMYCIN singularly achieves:

- far greater antibiotic activity with far less drug
- greater stability in body media
- unrelenting peak activity throughout therapy
- "extra-day" protection through sustained activity

DECLOMYCIN retains:

- unsurpassed broad-spectrum range of activity
- rapid activity
- excellent toleration
- effectiveness against infection in nearly all organs or systems—rapid diffusion in body tissues and fluids

**Chlortetracycline Lederle* †*Tetracycline Lederle*

DECLO

Demethylchlortetracycline Lederle

Far greater antibiotic activity with far less antibiotic

Milligram for Milligram, DECLOMYCIN exhibits 2 to 4 times the clinical potency (inhibitory action) of tetracycline against susceptible organisms. Thus, DECLOMYCIN has the advantage of providing significantly higher serum *activity* levels with significantly reduced drug intake.* [1,3,5]

Actually, DECLOMYCIN demonstrates the highest ratio of prolonged activity level to daily milligram intake of any known broad-spectrum antibiotic. Reduction of milligram intake of drug reduces hazards of related physical effect on intestinal mucosa or interaction with gastrointestinal contents.

*Activity level is a far more meaningful basis of comparison than quantitative blood levels, as Hirsch and Finland note. Action upon pathogens is the ultimate value.[1]

MYCIN

Unrelenting peak antimicrobial attack throughout therapy

The high level of DECLOMYCIN activity is uniquely sustained. It is not just an initial phenomenon but is constant—maintained on each day of treatment and between doses—without noticeable diminution of intensity. Peak-and-valley control is eliminated, favoring continuous suppression of pathogens and consequent improvement.

This DECLOMYCIN constant is achieved through remarkably greater stability in body fluids,[2,4,6] resistance to degradation[6] and a low rate of renal clearance[4,5]—all supporting antibiotic activity for extended periods.

DECLO

Demethylchlortetracycline Lederle

"Extra-day" activity for security against relapse

DECLOMYCIN maintains significant antibacterial activity for one to two days after discontinuance of dosage[1]—a major distinction from other antibiotics. Previous drugs have declined abruptly in activity following withdrawal.

DECLOMYCIN thus gives the patient an unusual degree of protection against resurgence of the primary infection, and against secondary infection... sequelae not infrequently encountered and often resembling a "resistance problem." Consequently, reinstitution of therapy or a change in therapy should rarely be necessary.

MYCIN

A masterpiece of

greater antibiotic activity

with far less antibiotic intake

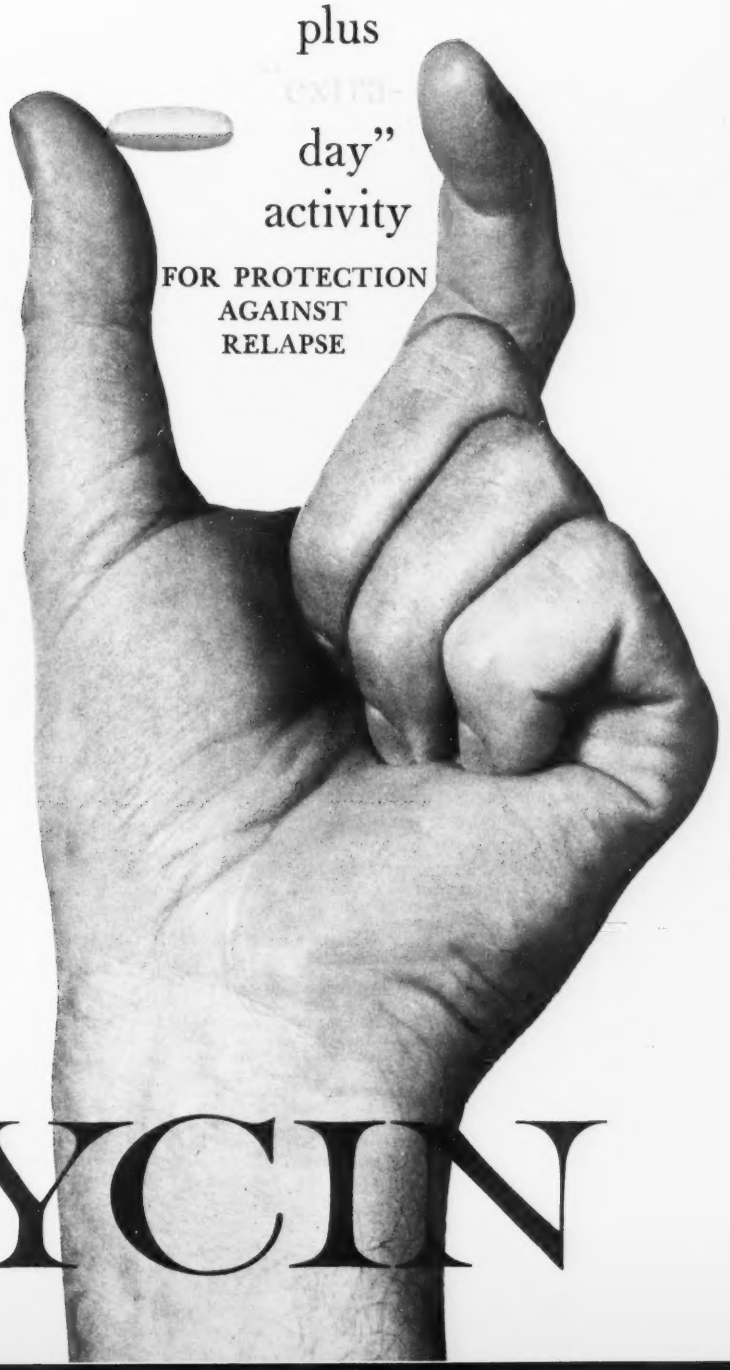
unrelenting peak attack

—enhancing the unsurpassed features of tetracycline... for greater physician-patient benefits

DECLO

Demethylchlortetracycline Lederle

antibiotic design



MYCIN

A major contribution of Lederle research

in the distinctive dry-filled duotone capsule

DECLOMYCIN®

Demethylchlortetracycline Lederle

available as:

DECLOMYCIN Capsules, 150 mg.
Adult dosage: 1 capsule four times daily.
Pediatric Drops, 60 mg. per cc.
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1. Hirsch, H. A., and Finland, M.: Antibacterial Activity Of Serum Of Normal Subjects After Oral Doses of Demethylchlortetracycline, Chlortetracycline and Oxytetracycline. *New England J. Med.* 260:1099 (May 28) 1959. 2. Hirsch, H. A., Kunin, C. M., and Finland, M.: Demethylchlortetracycline – A New And More Stable Tetracycline Antibiotic That Yields Greater and More Sustained Antibacterial Activity. To be published. 3. Lichter, E. A., and Sobel, S.: The Distribution Of Oral Demethylchlortetracycline In Healthy Volunteers And In Patients Under Treatment For Various Infections. To be published. 4. Kunin, C. M., Dornbush, A. C. and Finland, M.: Distribution And Excretion Of Four Tetracycline Analogues In Normal Young Men. To be published. 5. Kunin, C. M., and Finland, M.: Demethylchlortetracycline: New Tetracycline Antibiotic That Yields Greater and More Sustained Antibacterial Capacity. *New England J. Med.* 259:999 (Nov. 28) 1958. 6. Sweeney, W. M.; Hardy, S. M.; Dornbush, A. C., and Ruegsegger, J. M.: Demethylchlortetracycline: A Clinical Comparison of A New Antibiotic with Chlortetracycline and Tetracycline. *Antibiotics & Chemotherapy* 9:13 (Jan.) 1959.

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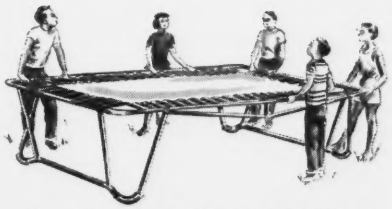

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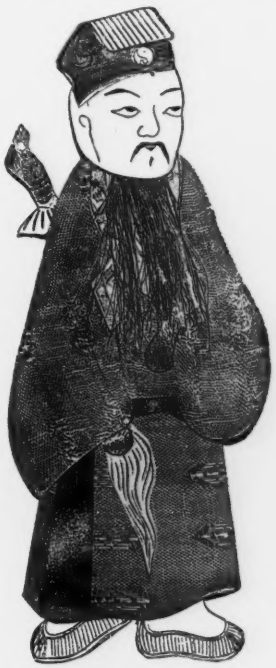

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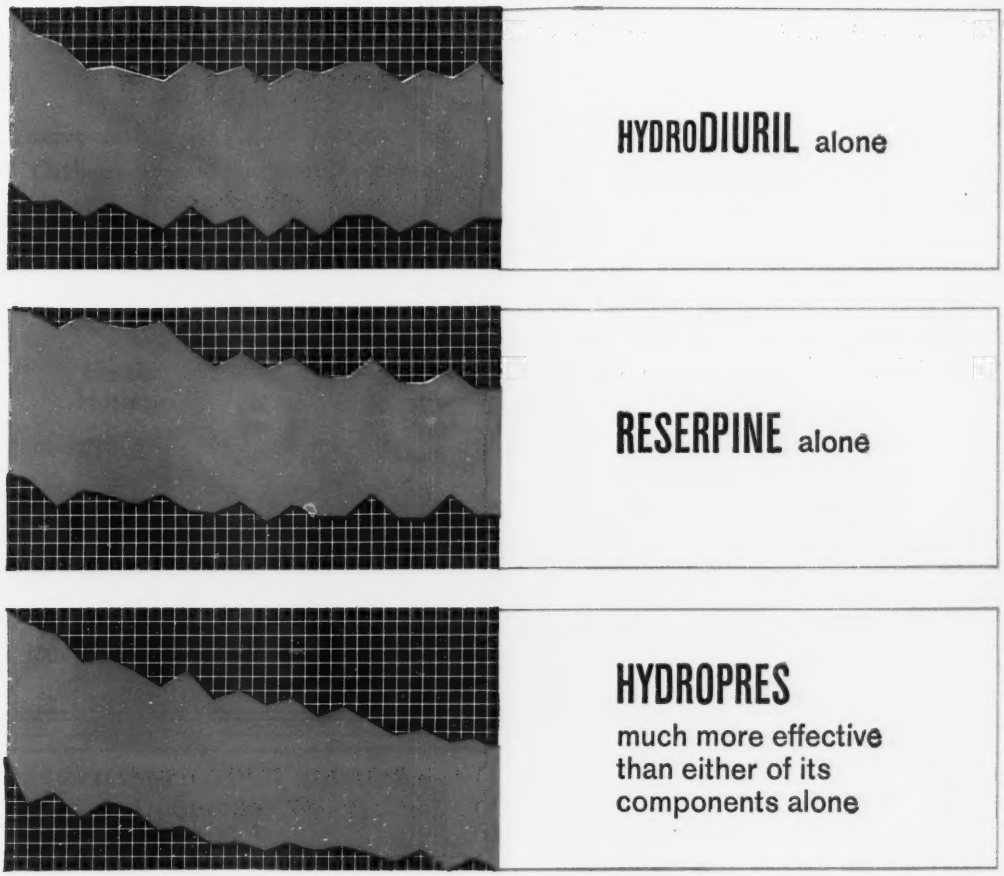

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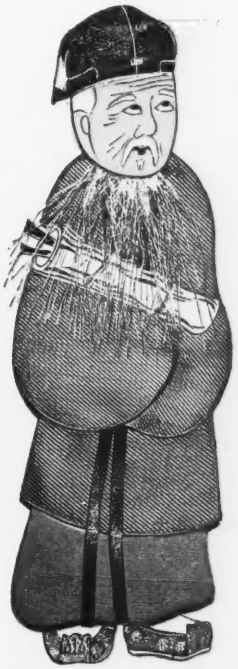

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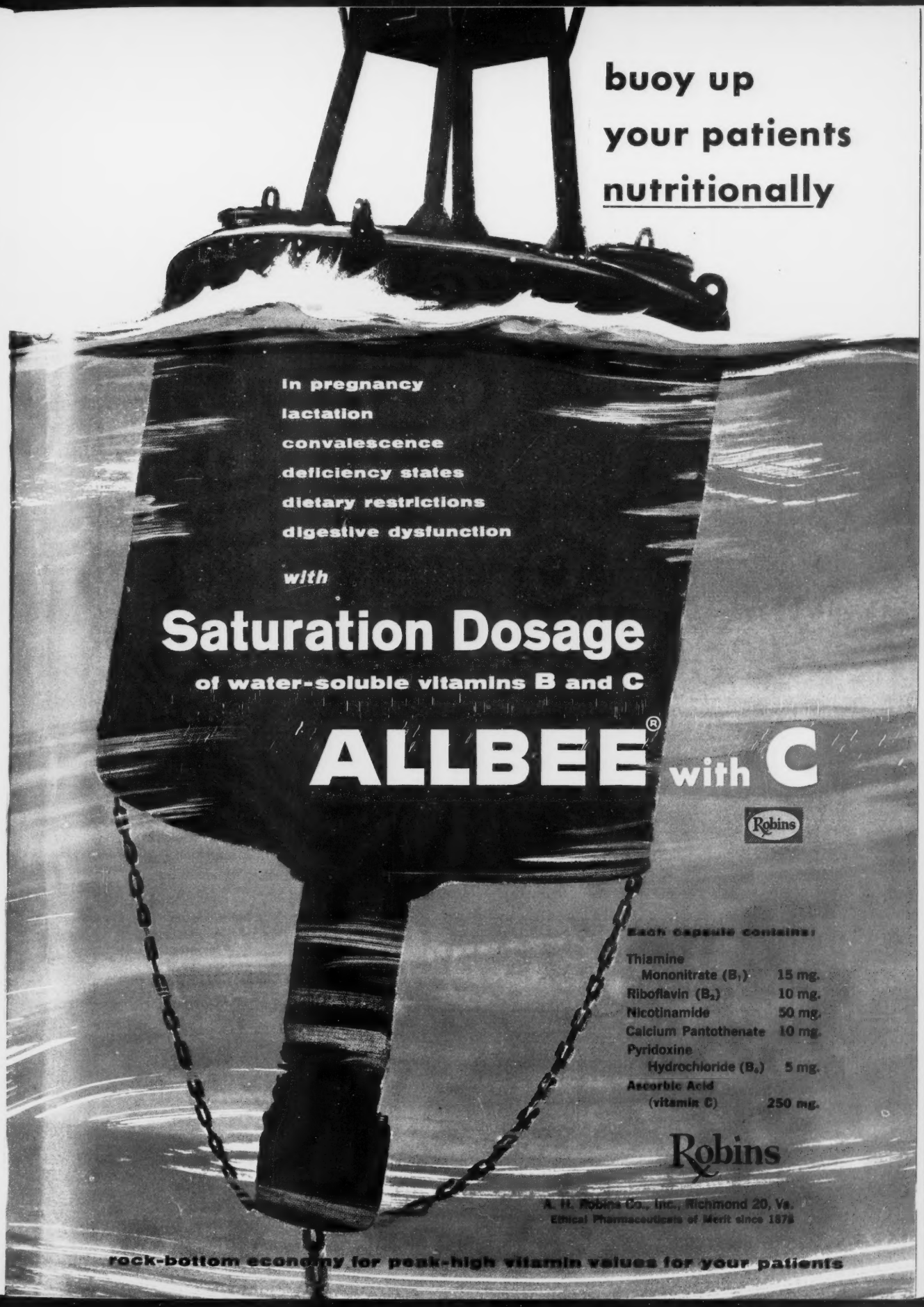
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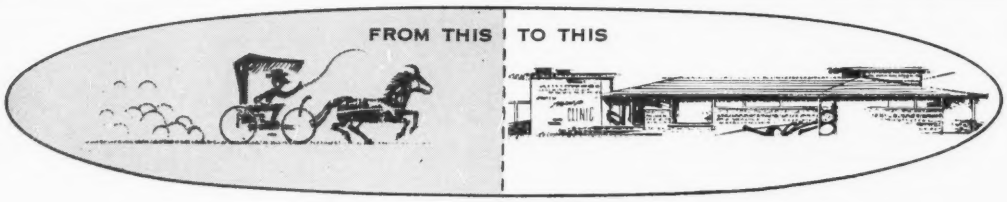

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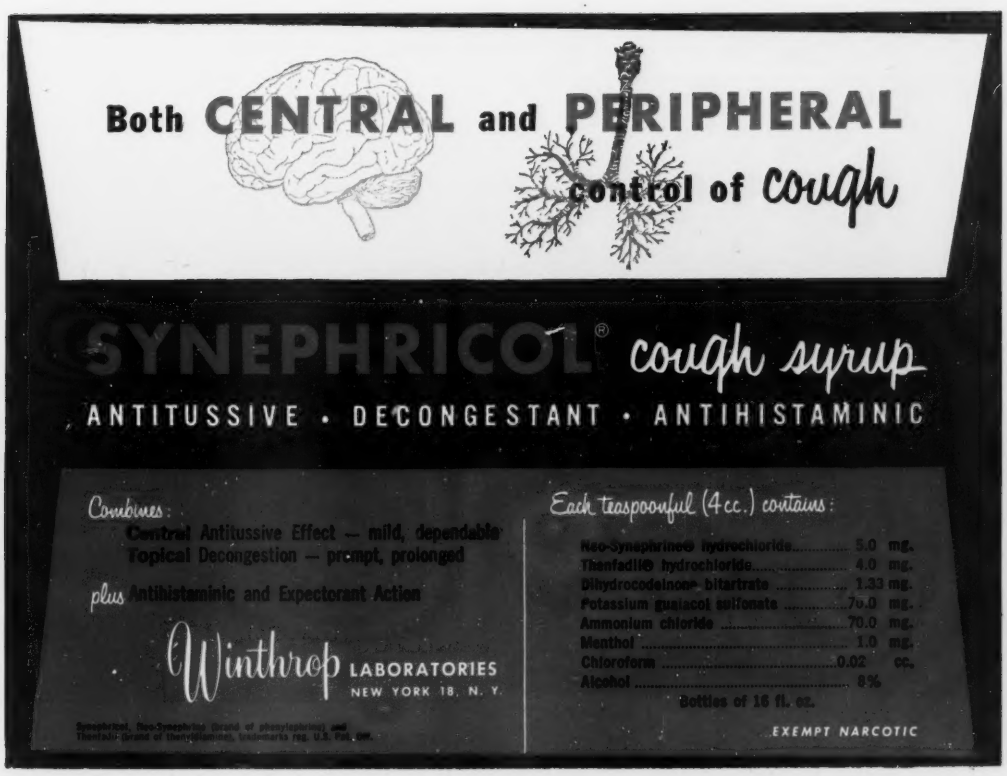

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